passmedicine PART 2

INFECTIOUS DES

2024

A 44-year-old man is brought to the emergency department by his wife with an acute onset of confusion. She reports that her husband had only just recently recovered from a bout of nausea and vomiting for the last 10 days since returning home from visiting family in Vietnam. He has no past medical history of note and takes no regular medications. His sister was treated for tuberculosis 6 months ago.

His observations are as follows:

- Temperature 38.3ºC
- Heart rate 116 beats/min
- Blood pressure 112/76 mmHg
- Respiratory rate 16 breaths/min
- Saturations 96% on air

On examination, he is disoriented to time and place but recognises his wife. He complains of a severe headache but the remainder of his neurological examination is unremarkable. On auscultation, his chest is clear and heart sounds are normal. His abdomen is soft and non-tender.

What is the most likely diagnosis?

- Dengue fever
- Disseminated tuberculosis
- Japanese encephalitis
- Meningitis
- Yellow fever

Submit answer

Reference ranges

Score: **0%**

1 -

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What is the most likely diagnosis?

Dengue fever	11%
Disseminated tuberculosis	3%
Japanese encephalitis	72%
Meningitis	5%
Yellow fever	8%

Confusion in returned traveller from Asia -> Japanese encephalitis

Important for me Less important

AI

This patient has **Japanese encephalitis**. Spread by Culex mosquitoes that breed in rice paddy fields, Japanese encephalitis is the most common cause of encephalitis in South East Asia, China and India. Most cases are asymptomatic. However, in more severe cases, symptoms include headache, fever, seizures and confusion. A prodrome of nausea and vomiting may be seen initially, as described by this patient. This patient is presenting with acute confusion and headache

following a trip to Vietnam and therefore, a diagnosis of Japanese encephalitis is likely.

Dengue fever is a viral illness that presents with fever, headache and myalgia. A maculopapular rash early on in the illness is common, along with facial flushing. In some patients, the disease can progress to a viral haemorrhagic fever and dengue shock syndrome. However, it does not present with symptoms of acute encephalitis.

Disseminated tuberculosis is a less likely answer. Typically tuberculosis affects the respiratory system and systemic symptoms are present including fever, night sweats and weight loss. Although his sister was recently treated for tuberculosis, the absence of these constitutional symptoms and the more abrupt onset of illness suggests an alternative diagnosis.

Meningitis can have a viral or bacterial cause. However, the presenting symptoms are similar and include fever, headache and meningism. Vomiting may also feature. However, the absence of photophobia and meningism and the acute onset of confusion makes a diagnosis of encephalitis more likely than meningitis.

Yellow fever is a viral haemorrhagic illness common in Central and South America. It presents as a mild flu-like illness followed by a brief remission period before the onset of haematemesis, jaundice and oliguria. The presenting symptoms in this question do not describe any form of haemorrhage and are not typical of yellow fever, suggesting that it is not the underlying diagnosis.

Discuss (3) | Improve

Next question ›

Japanese encephalitis ★

Japanese encephalitis is the most common cause of viral encephalitis in South East Asia, China the Western Pacific and India, with approx. 50,000 cases annually. It is a flavivirus transmitted by culex mosquitos which breeds in rice paddy fields. The reservoir hosts are aquatic birds, but pigs are an amplification host and therefore close domestic contact with pigs is a risk factor.

The majority of infection is asymptomatic.

Clinical features are headache, fever, seizures and confusion. Parkinsonian features indicate basal ganglia involvement. It can also present with acute flaccid paralysis.

Diagnosis is by serology or PCR. Management is supportive.

Prevention is a vaccine and there are a variety of different types.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **24%**

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A 34-year-old man presents to the emergency department with a three-week history of mild right upper quadrant (RUQ) pain. He feels sweaty and feverish. He reports a three-week holiday to Indonesia one-year ago.

His observations are:

- Respiratory rate: 19/min
- Oxygen saturation: 98% on room air
- Heart rate: 104bpm
- BP: 138/72mmHg
- Temperature: 38.0 ^{o}C
- GCS: 15/15

On examination, the patient is tender in the RUQ and appears sweaty. Otherwise, he examines normally.

An abdominal ultrasound is performed which shows a 7cm abscess in the right lobe of the liver. Stool microscopy shows spherical cysts.

What is the most appropriate treatment?

- Abscess aspiration
- Amoxicillin + ciprofloxacin + metronidazole
- Metronidazole for 1 week
- Metronidazole for 1 week, then diloxanide for 1 week
- Praziquantel

Submit answer

Reference ranges

Score: **0%**

1 -

2 -

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On examination, the patient is tender in the RUQ and appears sweaty. Otherwise, he examines normally.

An abdominal ultrasound is performed which shows a 7cm abscess in the right lobe of the liver. Stool microscopy shows spherical cysts.

What is the most appropriate treatment?

Abscess aspiration	18%
Amoxicillin + ciprofloxacin + metronidazole	23%
Metronidazole for 1 week	7%
Metronidazole for 1 week, then diloxanide for 1 week	42%
Praziquantel	10%

Treatment for invasive amoebiasis should be followed by a luminal amoebicide to eradicate the cystic stage which is resistant to metronidazole and tinidazole (which are used against the invasive stage)

Important for me Less important

Metronidazole for 1 week, then diloxanide for 1 week is the correct answer. This patient has amoebiasis, with the spherical cysts representing *Entamoeba histolytica* cysts. The incubation

period is from days to years. Amoebiasis has a worldwide distribution but is more common in tropical and subtropical regions. Metronidazole treats Entamoeba trophozoites (but has insufficient activity against cysts). Diloxanide or paromomycin are luminal agents that are active in the bowel - they treat the Entamoeba cysts but are poorly absorbed, and so are ineffective against organisms outside the bowel. Therefore a combination of medications is required.

Abscess aspiration is the incorrect answer. Indications for abscess aspiration in amoebiasis are:

- Abscesses in the left liver lobe (possible rupture into pericardium)
- Abscesses >10cm in diameter
- Severe pain
- Marked diaphragm elevation
- Abscesses that fail to respond to medical management
- Impending abscess rupture

Amoxicillin + ciprofloxacin + metronidazole is the incorrect answer. This is the recommended treatment for bacterial liver abscesses, but this will not treat amoebiasis. Patients with bacterial liver abscesses are likely to be significantly more unwell (septic). They will also not have the characteristic cysts or trophozoites on stool microscopy.

Metronidazole for 1 week is the incorrect answer. Metronidazole will treat the Entamoeba trophozoites, but not the cysts.

Praziquantel is the incorrect answer. Praziquantel is an anti-helminthic drug and will not treat *Entamoeba histolytica*, which is a protozoan. Praziquantel (in combination with percutaneous aspiration) can be used to treat hydatid liver cysts caused by *Echinococcus granulosus*. Hydatid cysts tend to be multi-loculated and asymptomatic.

Discuss (3) | Improve

Next question >

Amoebiasis

Amoebiasis is caused by *Entamoeba histolytica* (an amoeboid protozoan) and spread by the faecal-oral route. It is estimated that 10% of the world's population is chronically infected. Infection can be asymptomatic, cause mild diarrhoea or severe amoebic dysentery. Amoebiasis also causes liver and colonic abscesses.

Amoebic dysentery

- profuse, bloody diarrhoea
- there may be a long incubation period
- stool microscopy may show trophozoites if examined within 15 minutes or kept warm (known as a 'hot stool')

- treatment
  - oral metronidazole
  - a 'luminal agent' (to eliminate intraluminal cysts) is recommended usually as well e.g. diloxanide furoate

Amoebic liver abscess

- usually a single mass in the right lobe (may be multiple). The contents are often described as 'anchovy sauce'
- features
  - fever
  - right upper quadrant pain
  - systemic symptoms e.g. malaise
  - hepatomegaly
- investigations
  - ultrasound
  - serology is positive in > 95%
- management
  - oral metronidazole
  - a 'luminal agent' (to eliminate intraluminal cysts) is recommended usually as well e.g. diloxanide furoate



Next question

Textbooks

High-yield textbook

Extended textbook

Links

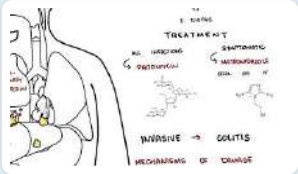
Patient.info

13 6

Amoebiasis review

Suggest link Report broken link

Media



Amebiasis - entamoeba histolytica symptoms, pathophysiology, diagnosis, treatment

Armando Hasudungan - YouTube 0 0

Report broken media

Score: **24%**

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A 54-year-old man presents to the emergency department with two days of pain and a rash on his right thigh. He has recently had the chickenpox for the first time and was given a course of aciclovir, which he has now completed. The lesions have now healed. He has type 2 diabetes and is on metformin. He smokes ten cigarettes daily.

His observations are heart rate 121 beats per minute, respiratory rate 24/minute, blood pressure 85/45 mmHg, oxygen saturations 97% on air and temperature 39.4ºC.

On examination, he appears unwell. He is diaphoretic and anxious. There is a tense erythematous area on his right thigh, which is severely painful. His pulse is bounding and there is a flow murmur heard in the aortic area.

Blood tests:

Hb	121 g/L	Male: (135-180) Female: (115 - 160)
Platelets	580 * 10^9/L	(150 - 400)
WBC	25.2 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	4.1 mmol/L	(3.5 - 5.0)
Urea	11.4 mmol/L	(2.0 - 7.0)
Creatinine	176 µmol/L	(55 - 120)
CRP	380 mg/L	(< 5)
Bilirubin	25 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	89 u/L	(3 - 40)
Î³GT	45 u/L	(8 - 60)
Albumin	29 g/L	(35 - 50)

Despite aggressive fluid resuscitation, his blood pressure remains 86/51 mmHg.

Given the likely diagnosis, what is the most important treatment?

- Hyperbaric oxygen therapy
- Intravenous flucloxacillin

- Intravenous immunoglobulin
- Surgery
- Tazocin and clindamycin

Submit answer

Reference ranges

Score: **0%**

1 -
2 -
3 -

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Î³GT	45 u/L	(8 - 60)
Albumin	29 g/L	(35 - 50)

Despite aggressive fluid resuscitation, his blood pressure remains 86/51 mmHg.

Given the likely diagnosis, what is the most important treatment?

Hyperbaric oxygen therapy	2%
Intravenous flucloxacillin	17%

Intravenous immunoglobulin	8%
Surgery	46%
Tazocin and clindamycin	27%

Chickenpox is a risk factor for invasive group A streptococcal soft tissue infections including necrotizing fasciitis

Important for me Less important

Surgery is the correct answer. This man has septic shock secondary to necrotizing fasciitis as evidenced by fever, hypotension, rash and pain out of proportion to the clinical findings. Furthermore, he has evidence of end-organ dysfunction (acute kidney injury). The most important treatment for necrotizing fasciitis is surgical debridement of infected tissue. All other treatments are considered adjunctive. Both recent chickenpox infection and diabetes are risk factors for this condition.

Hyperbaric oxygen therapy is incorrect. There have been case reports of this treatment being used for necrotizing fasciitis but it is not the mainstay of treatment.

Intravenous flucloxacillin is incorrect. This antibiotic would be a good choice for simple cellulitis but necrotizing fasciitis is a more severe infection, typically caused by gram-negative, gram-positive and anaerobic bacteria and therefore requires a broader antibiotic cover.

Intravenous immunoglobulin is incorrect. This can be used as adjunctive treatment to antibiotics and surgery in severe skin and soft tissue infections but is not initial therapy or most important.

Tazocin and clindamycin are incorrect. This is the correct antibiotic choice for necrotizing fasciitis as it gives a broad cover against aerobic and anaerobic organisms and clindamycin has the added benefit of inhibiting toxin production. However, the literature on necrotizing fasciitis is clear in regarding surgery as the most important intervention.

Discuss (1) Improve

Next question

Chickenpox

Chickenpox is caused by primary infection with varicella zoster virus. Shingles is a reactivation of the dormant virus in dorsal root ganglion

Chickenpox is highly infectious

- spread via the respiratory route
- can be caught from someone with shingles
- infectivity = 4 days before rash, until 5 days after the rash first appeared*
- incubation period = 10-21 days

Clinical features (tend to be more severe in older children/adults)

- fever initially
- itchy, rash starting on head/trunk before spreading. Initially macular then papular then vesicular
- systemic upset is usually mild

Management is supportive

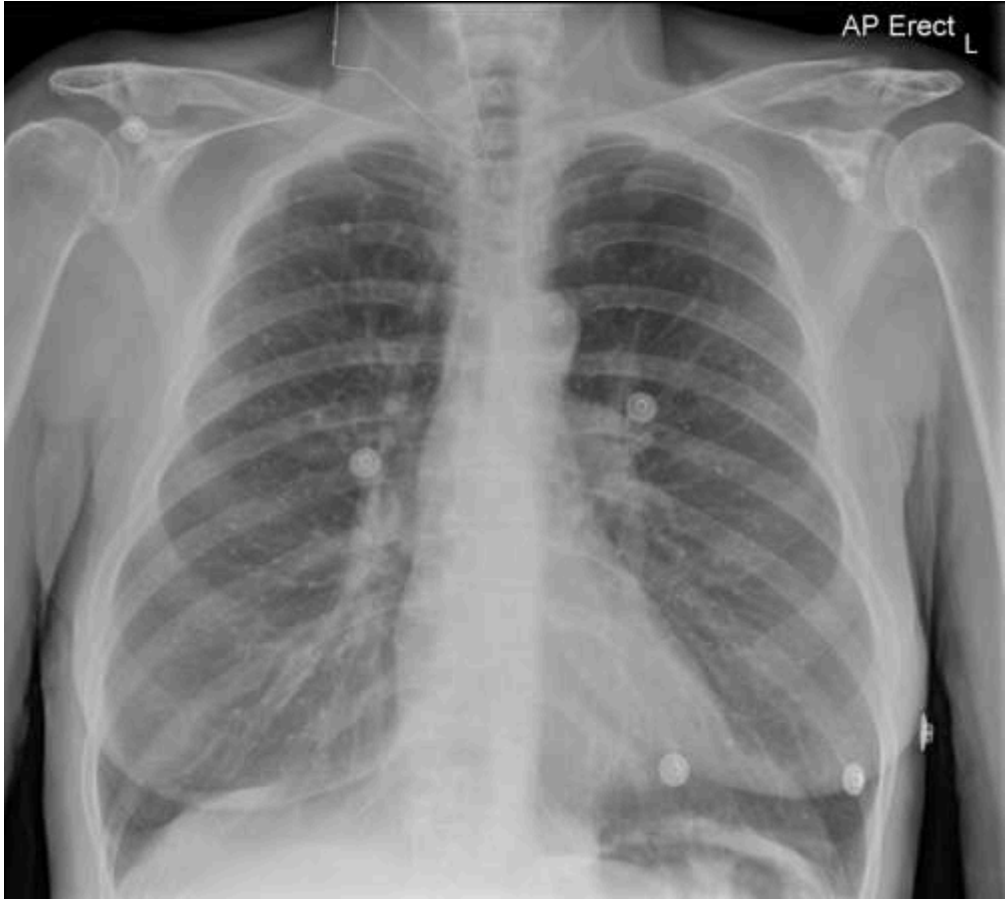
- keep cool, trim nails
- calamine lotion
- school exclusion: NICE state the following: *Advise that the most infectious period is 1-2 days before the rash appears, but infectivity continues until all the lesions are dry and have crusted over (usually about 5 days after the onset of the rash).*
- immunocompromised patients and newborns with peripartum exposure should receive varicella zoster immunoglobulin (VZIG). If chickenpox develops then IV aciclovir should be considered

A common complication is secondary bacterial infection of the lesions

- NSAIDs may increase this risk
- whilst this commonly may manifest as a single infected lesion/small area of cellulitis, in a small number of patients invasive group A streptococcal soft tissue infections may occur resulting in necrotizing fasciitis

Rare complications include

- pneumonia
- encephalitis (cerebellar involvement may be seen)
- disseminated haemorrhagic chickenpox
- arthritis, nephritis and pancreatitis may very rarely be seen



© Image used on license from Radiopaedia

Chest x-ray showing miliary opacities secondary to healed varicella pneumonia. Multiple tiny calcific miliary opacities noted throughout both lungs. These are of uniform size and dense suggesting calcification. There is no focal lung parenchymal mass or cavitating lesion seen. The appearances are characteristic for healed varicella pneumonia.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Greenbook 8 6

Varicella

Public Health England 9 6

Chapter 9: managing specific infectious diseases

Suggest link Report broken link

Media



Varicella zoster virus

Osmosis - YouTube 6 0

Report broken media

Score: **24%**

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A 31 year-old male originally from Russia who is known to be HIV positive presents with a rash. On examination there are purple-red cutaneous macules and papules on the back, neck and oral mucosal membrane.

Blood results reveal:

Hb	141 g/l
Platelets	327 * 10^9/l
WBC	4.2 * 10^9/l
Na^+	141 mmol/l
K^+	3.7 mmol/l
Urea	4.6 mmol/l
Creatinine	59 µmol/l
Bilirubin	27 µmol/l
ALP	97 u/l
ALT	44 u/l
Albumin	30 g/l
HIV viral load	14,000 copies / ml
CD4 cell count	124 cell/mm^3

A skin biopsy is performed and the report is below:

Spindle cells present. Mitotic activity is moderate. Abnormally dense and irregular blood vessels. Intracellular hyaline bodies present.

Which virus is likely to be responsible for these lesions?

- CMV
- EBV
- HIV-2
- HHV-8

HPV-8

Submit answer

Reference ranges

Score: **0%**

1 -
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4 -

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Spindle cells present. Mitotic activity is moderate. Abnormally dense and irregular blood vessels. Intracellular hyaline bodies present.

Which virus is likely to be responsible for these lesions?

CMV	8%
EBV	5%
HIV-2	1%
HHV-8	76%

HPV-8 9%

Kaposi's sarcoma - caused by HHV-8 (human herpes virus 8)

Important for me Less important

This is a description of AIDS-related Kaposi's sarcoma.

Kaposi sarcoma is a disease of blood vessels that was considered very rare before the start of the AIDS pandemic. AIDS is due to infection with human immunodeficiency virus (HIV).

There are four types of Kaposi sarcoma:

- The classic type of Kaposi sarcoma affects elderly men of Mediterranean and Middle European descent and in men in Sub-Saharan Africa.
- AIDS-associated Kaposi sarcoma.
- Endemic or African Kaposi sarcoma arises in some parts of Africa in children and young adults.
- Iatrogenic Kaposi sarcoma is due to drug treatment causing immune suppression.

Classic Kaposi sarcoma is rare and unassociated with HIV infection. It most often arises in middle-aged to elderly men of Mediterranean or Jewish descent (less than 10% are women), particularly if they come from a rural environment. They have a higher than expected rate of diabetes mellitus.

AIDS-related Kaposi sarcoma, unlike other forms of the disease, tends to have an aggressive clinical course. It is the most common presentation of Kaposi sarcoma. It has become less common in the US and Europe because of effective HAART treatment for HIV disease.

Kaposi's sarcoma is caused by infection with human herpesvirus 8 (HHV8), also known as Kaposi sarcoma-associated herpesvirus (KSHV) or KS agent

Typical histologic findings include proliferation of spindle cells, prominent slit-like vascular spaces, intracellular hyaline bodies and extravasated red blood cells.

Discuss (1) Improve

Next question

HIV: Kaposi's sarcoma

Kaposi's sarcoma

- caused by HHV-8 (human herpes virus 8)

- presents as purple papules or plaques on the skin or mucosa (e.g. gastrointestinal and respiratory tract)
- skin lesions may later ulcerate
- respiratory involvement may cause massive haemoptysis and pleural effusion
- radiotherapy + resection



Kaposi's sarcoma in a patient with HIV

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermIS.net

9 6

Picture of Kaposi's sarcoma

DermNet NZ

4 1

Kaposi's sarcoma

Patient.info 6 0

Kaposi's sarcoma

Suggest link Report broken link

Media



Human herpesvirus 8 (Kaposi's sarcoma)

Osmosis - YouTube 2 0



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube 1 0



Kaposi's sarcoma

Townsend Teaching - YouTube 1 0

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Score: **24%**

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A 24-year-old man presents to the emergency department with a sudden onset of fever, headache and malaise, in association with diarrhoea and cramping abdominal pain. He has no past medical history and is on no regular medications. He returned from the 'python cave' in the Maramagambo Forest in western Uganda six days previously. He had visited the cave to view the colonies of Egyptian fruit bats. He tells you he strictly followed precautions to prevent mosquito bites and took malaria prophylaxis for the duration of his time there.

His observations are heart rate 120 beats per minute, blood pressure 83/59 mmHg, respiratory rate 21/minute, oxygen saturations 95% on room air and temperature 39.4ºC.

On examination, he is confused with a Glasgow coma scale of 14/15 and is mildly jaundiced. There is no meningism or nuchal rigidity. His abdomen is moderately tender without evidence of peritonism. Chest auscultation is normal. His capillary refill time is 4 seconds, he has dry mucus membranes and decreased skin turgor. His heart sounds are normal with a soft systolic flow murmur audible.

Intravenous access is obtained and it is noted that he is oozing blood from his cannulae sites and also from his oral mucosa.

Urinalysis is unremarkable.

An ECG demonstrates sinus tachycardia.

Blood tests:

Hb	98 g/L	Male: (135-180) Female: (115 - 160)
Platelets	487 * 10^9/L	(150 - 400)
WBC	3.8 * 10^9/L	(4.0 - 11.0)
Na^+	131 mmol/L	(135 - 145)
K^+	5.1 mmol/L	(3.5 - 5.0)
Urea	13.1 mmol/L	(2.0 - 7.0)
Creatinine	280 µmol/L	(55 - 120)
CRP	50 mg/L	(< 5)
Bilirubin	84 µmol/L	(3 - 17)
ALP	267 u/L	(30 - 100)
ALT	432 u/L	(3 - 40)

Î³GT	180 u/L	(8 - 60)
Albumin	28 g/L	(35 - 50)
Prothrombin time	18.4 seconds	(11-14)

Plain radiography of the chest is normal.

Three thick and thin blood films are negative.

What is the most likely diagnosis?

- Hepatitis A
- Leptospirosis
- Malaria
- Marburg virus
- Typhoid

Submit answer

Reference ranges

Score: **0%**

1 -
2 -
3 -
4 -
5 -

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Exposure to bats/caves is a risk factor for Marburg virus, which results in a viral haemorrhagic fever

Important for me Less important

Marburg virus is the correct answer. The patient has been to a specific region in Uganda that has previously been associated with outbreaks of Marburg virus. Exposure to bats and caves is also a risk factor for this viral hemorrhagic fever. This condition typically presents with an abrupt onset of fever, headache and malaise and progresses to include gastrointestinal symptoms, arthralgia/myalgia and subsequently haemorrhagic complications with multi-organ dysfunction as evidenced in this case. The incubation period before the onset of symptoms is typically 3-10 days. In addition to the geographic and zoonotic risk factors, it is the only diagnosis listed that could account for the haemorrhagic complications in this scenario (oozing from venepuncture sites and bleeding from mucosal membranes). Treatment is supportive and the condition is associated with a high mortality rate.

Hepatitis A is incorrect. While this could account for jaundice and transaminitis, it is typically a self-limiting illness and associated with fulminant liver failure only in very rare cases. It is not associated with haemorrhagic complications.

Leptospirosis is incorrect. While this condition can present with fever, headache and liver dysfunction, the traditional vectors for transmission are mice, rats, cows and pigs. It is transmitted

classically by coming into contact with infected urine. While it is associated with subconjunctival haemorrhage, it is not usually associated with more diffuse haemorrhage as seen in this case. It is treated with antibiotics.

Malaria is incorrect. While this is the most common cause of the fever in the returning traveller, the patient has taken malaria prophylaxis and has had negative testing for malaria in addition to experiencing haemorrhagic complications, all of which make a diagnosis of malaria unlikely.

Typhoid is incorrect. While this is a common cause of fever in the returning traveller and is associated with fever and diarrhoea, it would not account for the haemorrhagic complications detailed in this scenario.

Discuss (8) | Improve

Next question >

Viral haemorrhagic fevers

Viral haemorrhagic fevers (VHFs) are a group of viral infections that result in presentations ranging from a flu-like illness to multisystem failure.

Examples of viral haemorrhagic fever (VHF) include:

- Flaviviridae: dengue, yellow fever
- Arenaviridae: Lassa fever
- Filoviridae: Ebola virus, Marburg virus
- Bunyaviridae: Hantaviruses, Crimean-Congo haemorrhagic fever, Rift Valley fever

Features

- flu-like symptoms
- abdominal pain
- haemorrhage
  - petechiae, bruising
  - bloody diarrhoea, haematemesis, haemoptysis
  - disseminated intravascular coagulation
- multiorgan failure

Prevention

- a yellow fever vaccine is available
- a dengue and Ebola vaccine have been developed but are now widely used at the current time
- no vaccines are available for the other VHFs

Management

- treatment is generally supportive
- ribavirin is used to treat Lassa fever

The more common VHFs such as dengue, Ebola and yellow fever are covered in more detail elsewhere in the notes.

Lassa fever

Lassa fever is contracted by contact with the excreta of infected African rats (Mastomys rodent) or by person-to-person spread.

Marburg virus

Causes a VHF very similar to Ebola.

Risk factors for transmissions include caves/bats and primates.



Next question

Textbooks

High-yield textbook

Extended textbook

Links

HPA

2 1

Management of viral haemorrhagic fever

Score: **24%**

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A 35-year-old man presents to the travel walk-in centre due to a week of diarrhoea following a recent holiday to Brazil. He had a stool sample sent for ova, cysts, and parasites which tested positive for *Strongyloides stercoralis*.

What medication should be prescribed for the patient?

- Azithromycin
- Diethylcarbamazine
- Ivermectin
- Praziquantel
- Triclabendazole

Submit answer

Reference ranges

Score: **0%**

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A 35-year-old man presents to the travel walk-in centre due to a week of diarrhoea following a recent holiday to Brazil. He had a stool sample sent for ova, cysts, and parasites which tested positive for *Strongyloides stercoralis*.

What medication should be prescribed for the patient?

Azithromycin	1%
Diethylcarbamazine	5%
Ivermectin	68%
Praziquantel	15%
Triclabendazole	11%

Ivermectin is the drug of choice for treatment of strongyloidiasis

Important for me Less important

Ivermectin is the drug of choice for strongyloidiasis which acts by deactivating chloride channels which result in paralysis and death of the parasite either directly or by causing the worms to starve.

Azithromycin is an effective antibiotic for *Typhoid*, *Campylobacter* and often treatment of choice for traveller's diarrhoea. It is not effective for treatment of *Strongyloides*.

Diethylcarbamazine is not used to treat Strongyloides. It is the treatment of choice for *Wuchereria bancrofti*, *Loa Loa*, *Toxocara canis*.

Praziquantel is also not an effective treatment for Strongyloides. It is used to treat *Schistosoma haematobium*, *Paragonimus westermani* and *Clonorchis sinensis*.

Triclabendazole is the treatment of choice for *Fasciola hepatica* (the liver fluke) and is not the treatment of choice for Strongyloides.

Next question

Strongyloides stercoralis

Strongyloides stercoralis is a human parasitic nematode worm. The larvae are present in soil and gain access to the body by penetrating the skin. Infection with *Strongyloides stercoralis* causes strongyloidiasis.

Features

- diarrhoea
- abdominal pain/bloating
- papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks
- larva currens: pruritic, linear, urticarial rash
- if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered

Treatment

- ivermectin and albendazole are used

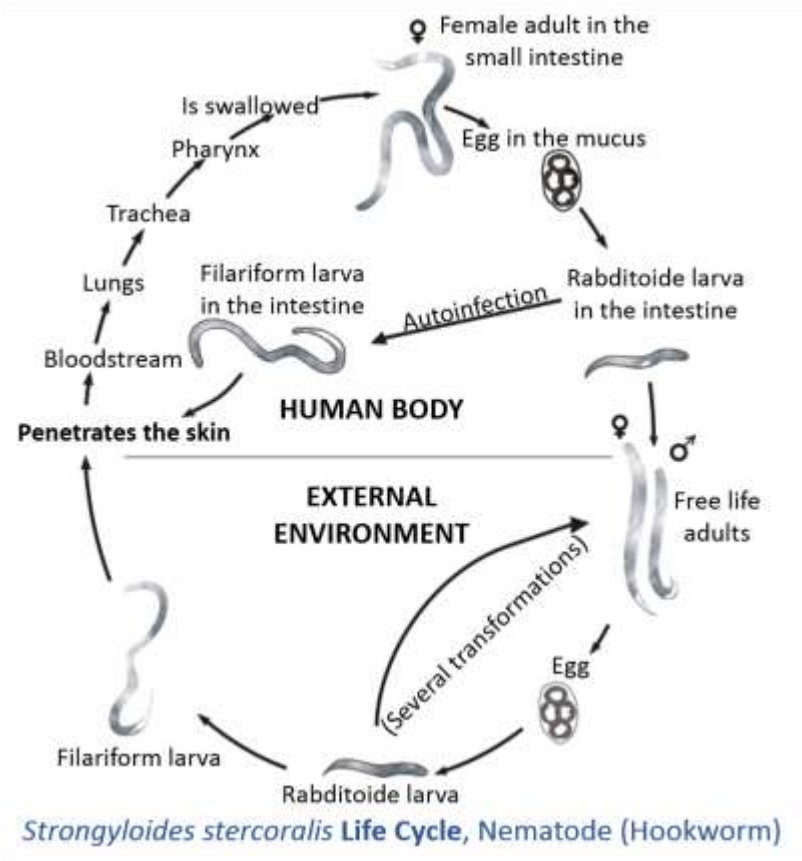


Image sourced from Wikipedia

Diagram showing the lifecycle of *Strongyloides stercoralis*

Next question

Textbooks

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Score: **24%**

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A 29-year-old man presented with fever, headache, a dry cough, and bloody diarrhoea. Symptoms started eight days ago. Eight weeks earlier he had been on an adventure holiday in Zambia with a group of friends, which involved rafting and visiting a game reserve. A couple of his friends had developed a similar illness. On examination, he was febrile, and was noted to have an urticarial rash on his trunk. He had a polyphonic wheeze in both lung fields and the tip of his spleen was palpable.

What is the pathogen causing the patient's symptoms?

Hb	134g/l (130 - 180)
Platelets	350 * 10^9/l (150 - 400)
WBC	12.2 * 10^9/l (4.0 - 11.0)
Neutrophil	6.5 * 10^9/l (1.5 - 7.0)
Lymphocyte	1.7 * 10^9/l (1.5 - 4.0
Eosinophil	3.4 * 10^9/l (0.04 - 0.4)
Monocyte	1.5 * 10^9/l (0<0.8)

- Dengue virus
- Epstein-Barr virus
- Human immunodeficiency virus
- *Leptospira icterohaemorrhagica*
- *Schistosoma mansoni*

Submit answer

Reference ranges

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2 -
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Eosinophil	3.4 * 10^9/l (0.04 - 0.4)
Monocyte	1.5 * 10^9/l (0<0.8)

Dengue virus	4%
Epstein-Barr virus	2%
Human immunodeficiency virus	1%
Leptospira icterohaemorrhagica	22%
Schistosoma mansoni	70%

Acute schistomiasis is usually associated with marked eosinophilia, cough, bloody diarrhoea, and splenomegaly

Important for me Less important

AI

The patient has a marked eosinophilia, and presents with systemic symptoms, cough, bloody diarrhoea, and splenomegaly eight weeks after exposure to fresh water in Africa. The most likely diagnosis in this scenario is Katayama syndrome, or acute schistosomiasis, caused by infection with

a small blood fluke. It most often occurs with Schistosoma japonicum and schistosoma mansoni infections but can occur with any species. Acute schistosomiasis should be treated with praziquantel 40-60 mg/kg as a single dose and short course of steroids to control the inflammatory reaction. The dose of praziquantel should be repeated after six weeks when flukes are fully mature because immature flukes are relatively resistant to treatment.

Eosinophilia is not a feature of glandular fever, acute dengue, EBV infection, or HIV. The incubation period is too long for leptospirosis and dengue, which typically is 3-14 days.

Discuss (4) | Improve

Next question >

Schistosomiasis

Schistosomiasis, or bilharzia, is a parasitic flatworm infection. The three main species of schistosome are *S. mansoni*, *S. japonicum* and *S. haematobium*.

Acute infections

Acute symptoms typically only develop in people who travel to endemic areas, as they don't have any immunity to the worms.

Acute manifestations may include:

- swimmers' itch
- acute schistosomiasis syndrome (Katayama fever)
  - fever
  - urticaria/angioedema
  - arthralgia/myalgia
  - cough
  - diarrhoea
  - eosinophilia

Chronic infections

Schistosoma haematobium

These worms deposit egg clusters (pseudopapillomas) in the bladder, causing inflammation. The calcification seen on x-ray is actually calcification of the egg clusters, not the bladder itself.

Depending on the site of these pseudopapillomas in the bladder, they can cause an obstructive uropathy and kidney damage.

This typically presents as a 'swimmer's itch' in patients who have recently returned from Africa. Schistosoma haematobium is a risk factor for squamous cell bladder cancer.

Features

- frequency
- haematuria
- bladder calcification

Investigation

- for asymptomatic patients serum schistosome antibodies are generally preferred
- for symptomatic patients the gold standard for diagnosis is urine or stool microscopy looking for eggs

Management

- single oral dose of praziquantel

Schistosoma mansoni and *Schistosoma japonicum*

These worms mature in the liver and then travel through the portal system to inhabit the distal colon. Their presence in the portal system can lead to progressive hepatomegaly and splenomegaly due to portal vein congestion.

These species can also lead to complications of liver cirrhosis, variceal disease and cor pulmonale.

Schistosoma intercalatum and *Schistosoma mekongi*

These are less prevalent than the other three forms, but are both attributed to intestinal schistosomiasis.



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 9 5

2011 Schistosomiasis review

Suggest link Report broken link

Media



Schistosomiasis (Bilharzia)â€“ an overview

Let's Learn About Bugs - YouTube 6 1

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Score: **24%**

1 ✓
2 ✗
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A 41-year-old man is admitted to the Emergency Department after having a seizure. He has no history of epilepsy but is known to be HIV positive. On examination he is still slightly confused post-ictally but there are no focal neurological signs. He is apyrexial and haemodynamically stable.

His current medications includes efavirenz, tenofovir, lamivudine and co-trimoxazole.

His latest CD4 count is 38 cells/μl.

CT scan (without contrast) is shown below:



© Image used on license from Radiopaedia

What is the most likely diagnosis?

- Cerebral toxoplasmosis
- Progressive multifocal leukoencephalopathy
- Cryptococcal infection
- CNS lymphoma
- Cerebral tuberculosis

Submit answer

Reference ranges

Score: **0%**

1 -
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His current medications includes efavirenz, tenofovir, lamivudine and co-trimoxazole.

His latest CD4 count is 38 cells/μl.

CT scan (without contrast) is shown below:



© Image used on license from Radiopaedia

What is the most likely diagnosis?

Cerebral toxoplasmosis	3%
Progressive multifocal leukoencephalopathy	5%
Cryptococcal infection	4%
CNS lymphoma	86%
Cerebral tuberculosis	2%

HIV, neuro symptoms, single brain lesions with homogenous enhancement - CNS lymphoma

Important for me Less important

A hyper-attenuating mass adjacent to the left lateral ventricle is seen. The homogenous appearance, position and history (low CD4 count and current use of co-trimoxazole prophylaxis) are suggestive of primary CNS lymphoma rather than cerebral toxoplasmosis.

Discuss Improve

Next question

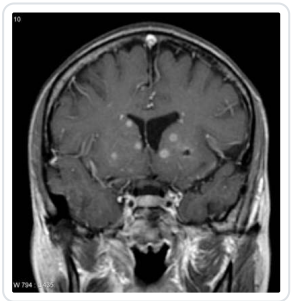
HIV: neurocomplications

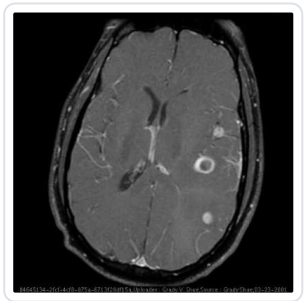
Focal neurological lesions

Toxoplasmosis

- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine



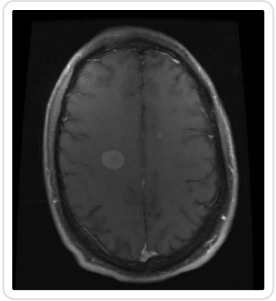




Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination

- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy



Next question

Textbooks

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Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link Report broken link

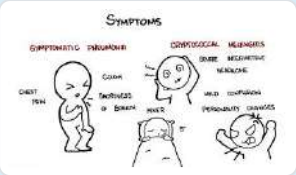
Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

0 1

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26 -

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A 32-year-old nurse presents to the the emergency department on the advise off occupational health. She contacted occupational health after she was coughed upon by a 25-year-old patient who is being treated with empirical antibiotics for bacterial meningitis. She felt contact on her face with droplets at the time. She has been feeling entirely well in herself. She has had appendicitis in the past as well as polycystic ovaries, but has no other medical problems. She takes metformin and oral contraception pills.

The 25-year-old patient was admitted 12 hours ago and has had a lumbar puncture which has confirmed meningococcal meningitis. What is the most appropriate plan for the nurse?

- Monitor for symptoms
- Lumbar puncture
- Give oral ciprofloxacin
- Admit to hospital for IV antibiotics
- Advise that the risk of transmission is not significant

Submit answer

Reference ranges

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The 25-year-old patient was admitted 12 hours ago and has had a lumbar puncture which has confirmed meningococcal meningitis. What is the most appropriate plan for the nurse?

Monitor for symptoms	4%
Lumbar puncture	0%
Give oral ciprofloxacin	86%
Admit to hospital for IV antibiotics	5%
Advise that the risk of transmission is not significant	5%

People who have been exposed to a patient with confirmed bacterial meningitis should be given prophylactic antibiotics if they been exposed to respiratory secretion, regardless of closeness of contact

Important for me Less important

This is a patient who has had a close contact with respiratory secretions of a person with confirmed meningococcal meningitis. Despite the fact that she has not had prolonged exposure, prophylaxis in the form of oral ciprofloxacin is needed.

The risk of transmission by this route is sufficiently significant to make monitoring for symptoms or not giving any treatment inappropriate.

A lumbar puncture is unlikely to be a helpful investigation for her as it cannot exclude that she will develop meningitis.

IV antibiotics are used to treat meningitis, whilst oral antibiotics can be used for prophylaxis of meningococcal meningitis.

Meningitis: management of suspected bacterial meningitis

The investigation and management of suspected bacterial meningitis are usually intertwined given the potential negative impact of delayed antibiotic treatment.

All patients should be transferred to hospital urgently. If patients are in a pre-hospital setting (for example a GP surgery) and meningococcal disease is suspected then intramuscular benzylpenicillin may be given, as long as this doesn't delay transit to hospital.

An ABC approach should be taken initially

- Airway
- Breathing
- Circulation
- Disability: GCS, ; focal neurological signs; seizures; papilloedema;

Senior review if any **warning signs** are present (please see the Meningitis Research Foundation algorithm link for the full list). Examples include:

- rapidly progressive rash
- poor peripheral perfusion
- respiratory rate < 8 or > 30 / min *or* pulse rate < 40 or > 140 / min
- pH < 7.3 *or* WBC< 4 *10^9/L *or* lactate > 4 mmol/L
- GCS < 12 or a drop of 2 points
- poor response to fluid resuscitation

A key decision is when/whether to attempt a lumbar puncture (LP)

- LPs require an aseptic technique and are potentially technical difficult - this means they make take time and delay other treatment
- on the other hand, having a cerebrospinal fluid (CSF) sample can be very useful in confirming the diagnosis and guiding antibiotic treatment
- as always, patient safety means that if there is any doubt IV antibiotics should be given as a priority

Lumbar puncture should be delayed in the following circumstances

- signs of severe sepsis or a rapidly evolving rash
- severe respiratory/cardiac compromise
- significant bleeding risk
- signs of raised intracranial pressure
    - focal neurological signs

    - papilloedema
    - continuous or uncontrolled seizures
    - GCS ≤ 12

Management of patients without indication for delayed LP

- IV access → take bloods and blood cultures
- lumbar puncture
    - if this cannot be done within the first hour, IV antibiotics should be given after blood cultures have been taken
- IV antibiotics
    - 3 months - 50 years: BNF recommends cefotaxime (or ceftriaxone)
    - > 50 years: BNF recommends cefotaxime (or ceftriaxone) + amoxicillin (or ampicillin) for adults
- IV dexamethasone
    - the BNF recommend to '*consider adjunctive treatment with dexamethasone (particularly if pneumococcal meningitis suspected in adults), preferably starting before or with first dose of antibacterial, but no later than 12 hours after starting antibacterial; avoid dexamethasone in septic shock, meningococcal septicaemia, or if immunocompromised, or in meningitis following surgery*'
- CT scan is not normally indicated

Management of patients with signs of raised intracranial pressure

- get critical care input
- secure airway + high-flow oxygen
- IV access → take bloods and blood cultures
- IV dexamethasone
- IV antibiotics as above
- arrange neuroimaging

Management of patients with signs of severe sepsis or a rapidly evolving rash

- get critical care input
- secure airway + high-flow oxygen
- IV access → take bloods and blood cultures
- IV fluid resuscitation
- IV antibiotics as above

Investigations

The bloods that have already been taken should be tested for the following:

- full blood count
- renal function
- glucose
- lactate

- clotting profile
- CRP

If an LP has been performed, the CSF should be tested for:

- glucose, protein, microscopy and culture
- lactate
- meningococcal and pneumococcal PCR
- enteroviral, herpes simplex and varicella-zoster PCR
- consider investigations for TB meningitis

Additional tests that may be helpful include:

- blood gases
- throat swab for meningococcal culture

BNF antibiotic guidelines

Scenario	BNF recommendation
Initial empirical therapy aged < 3 months	IV cefotaxime + amoxicillin (or ampicillin)
Initial empirical therapy aged 3 months - 50 years	IV cefotaxime (or ceftriaxone)
Initial empirical therapy aged > 50 years	IV cefotaxime (or ceftriaxone) + amoxicillin (or ampicillin)
Meningococcal meningitis	IV benzylpenicillin or cefotaxime (or ceftriaxone)
Pneumococcal meningitis	IV cefotaxime (or ceftriaxone)
Meningitis caused by *Haemophilus influenzae*	IV cefotaxime (or ceftriaxone)
Meningitis caused by Listeria	IV amoxicillin (or ampicillin) + gentamicin

If the patient has a history of immediate hypersensitivity reaction to penicillin or to cephalosporins the BNF recommends using chloramphenicol.

Management of contacts

Prophylaxis needs to be offered to households and close contacts of patients affected with meningococcal meningitis. Prophylaxis should also be offered to people who have been exposed to respiratory secretion, regardless of the closeness of contact. The risk to contacts is highest in the first 7 days but persists for at least 4 weeks.

Management

- people who have been exposed to a patient with confirmed bacterial meningitis should be given prophylactic antibiotics if they have close contact within the 7 days before onset
- oral ciprofloxacin or rifampicin may be used. The Health Protection Agency (HPA) guidelines now state that whilst either may be used ciprofloxacin is the drug of choice as it is widely available and only requires one dose
- meningococcal vaccination should be offered to close contacts when serotype results are available, including booster doses to those who had the vaccine in infancy
- for pneumococcal meningitis, no prophylaxis is generally needed. There are however exceptions to this. If a cluster of cases of pneumococcal meningitis occurs the HPA have a protocol for offering close contacts antibiotic prophylaxis. Please see the link for more details

AI + Q 123

Next question >

B I A T↕

Textbooks

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Links

Meningitis Research Foundation 22 13

Meningitis protocol

BNF 15 9

Medical emergencies in the community

Health Protection Agency 15 7

2018 Guidance for public health management of meningococcal disease in the UK

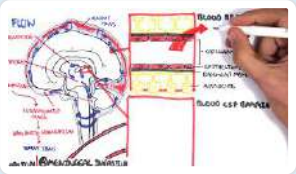
NICE

13 5

2010 Bacterial meningitis and meningococcal septicaemia guidelines

Suggest link Report broken link

Media



(Bacterial) Meningitis Pathophysiology

Armando Hasudungan - YouTube 5 0



Meningitis

Osmosis - YouTube 4 0

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1 ✓
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A 35-year-old woman presents to the ambulatory medical care unit with a 6-week history of an ulcer on the back of her right hand. It started as a 1cm painful nodule and gradually enlarged. It is now 3cm in diameter and is a firm raised purple/red lesion with some central ulceration. There are no lesions anywhere else and she is systemically well. She has no known past medical history and has not travelled outside of the UK in the last 5 years. She has no pets but works in a pet shop which specialises in tropical fish.

What organism is the most likely cause of this lesion?

- Herpes simplex virus
- Leishmania braziliensis
- Mycobacterium marinum
- Mycobacterium ulcerans
- Sporothrix schenckii

Submit answer

Reference ranges

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What organism is the most likely cause of this lesion?

Herpes simplex virus	1%
Leishmania braziliensis	11%
Mycobacterium marinum	77%
Mycobacterium ulcerans	4%
Sporothrix schenckii	6%

Fish tank granuloma is caused by *Mycobacterium marinum*

Important for me Less important

AI

This presentation is typical of fish tank granuloma, which is caused by *Mycobacterium marinum*. The main clue is her exposure to fish tanks and lack of risk factors for the other organisms. As well as via contact of skin with the water in fish tanks or aquariums, the infection can less commonly be acquired through preparing fish or shellfish, or contact with fishing equipment. It generally presents as a slow-growing lesion, although it may disseminate or progress more quickly in immune-suppressed people. *Mycobacterium marinum* grows most effectively at 30 degrees, and so the lesions are usually found at the extremities.

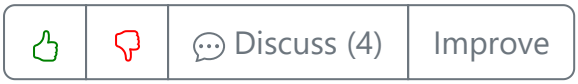
Herpes simplex virus can cause lesions on the hand if the skin of the hand comes into contact with infected skin around the mouth or genitals - this is called herpetic whitlow. However, the lesion would usually be on the fingertips rather than the back of the hand, would start with vesicles rather than a nodule, and would generally progress more quickly.

Leishmania braziliensis causes cutaneous and mucocutaneous leishmaniasis and is found in Central and South America. Cutaneous leishmaniasis may present similarly to fish tank granuloma,

however, the patient has not travelled abroad.

Mycobacterium ulcerans causes painful ulcers on the skin which are often called Buruli ulcers - however, it is found predominantly in tropical regions.

Sporothrix schenckii: is a dimorphic fungus which can cause similar lesions on the hands and arms after contact with infected plant matter - it is often referred to as rose gardener's disease. There tend to be several lesions which spread up the lymphatic system.



Next question

Mycobacterium marinum

Mycobacterium marinum is one of many mycobacteria that can cause disease in humans. Fish tank granuloma typically presents in patients who have had an exposure to, or frequently work with fish. It has an incubation period of 3-4 weeks and lesions can be painful or painless. A cut or break in the skin can be enough for the organism to enter the blood stream and track up the lymphatic system (sporotrichoid spread). Treatment options include tetracyclines, fluoroquinolones, sulfonamides and macrolides.



Next question



Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

4 0

Atypical mycobacterial infection

Suggest link Report broken link

Score: **24%**

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A 23 year-old student presents with a febrile illness, seven days after return from holiday in Thailand. He delayed seeking medical attention because he thought it was 'just a virus', and he had experienced similar self-limiting symptoms when travelling to Vietnam several years earlier. He did not take malaria prophylaxis due to a previous adverse reaction to mefloquine. The illness began two days after his return, when he recorded his temperature at 38.4°C. He also complains of severe generalised joint pains and headache which he describes as 'behind the eyes'. Today he has developed nosebleeds and when he brushed his teeth this morning he noticed that his gums bled.

On examination his temperature is 38.2°C and there is a widespread macular rash with islands of sparing. There are some petechial haemorrhages on the limbs. Cardio-respiratory examination is unremarkable except for tachycardia of 114. Blood pressure is 123/79 mmHg. On palpation of the abdomen you discover tender hepatomegaly. Neurological examination is unremarkable.

On application of a tourniquet to take a blood sample you notice that the area under the tourniquet has become bruised.

Results of blood tests are as follows:

Hb	14.1 g/dl
MCV	94.2 fl
Haematocrit	0.57
Platelets	23 x10^9/l
WCC	6.1 x10^9/l

Na^+	134 mmol/l
K^+	4.6 mmol/l
Urea	3.8 mmol/l
Creatinine	80 µmol/l

ALT	64 IU/l
ALP	78 IU/l
Bilirubin	13 mol/l
Albumin	28 g/l

Rapid Malaria test	Negative

Thick and thin blood films	Pending

What is the most likely diagnosis?

- Non-falciparum malaria
- Dengue haemorrhagic fever
- Lassa fever
- Meningococcal septicaemia
- HIV seroconversion

Submit answer

Reference ranges

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Bilirubin	13 mol/l
Albumin	28 g/l

Rapid Malaria test	Negative

Thick and thin blood films	Pending

What is the most likely diagnosis?

Non-falciparum malaria	2%
Dengue haemorrhagic fever	**94%**
Lassa fever	3%
Meningococcal septicaemia	1%
HIV seroconversion	0%

Retro-orbital headache, fever, facial flushing, rash, thrombocytopenia in returning traveller → ?dengue

Important for me Less important

This is a viral haemorrhagic fever, and the clinical picture is classic for dengue. The disease is endemic in South-East Asia. A defective immune response in secondary infections means that individuals who have been infected with one viral serotype may develop more severe disease when later infected with a different serotype. This patient may have developed self-limiting dengue fever on a previous trip to Vietnam, and now has secondary infection.

Dengue fever typically begins 7-10 days after infection with a rapid onset of fever, severe myalgia, and retro-orbital headache. A maculopapular rash with islands of sparing is characteristic. In the majority of cases there is self-resolution after 2-7 days. In a proportion of cases however, especially in secondary infection, the disease progresses to a more fulminant form known as dengue haemorrhagic fever. There is widespread vascular breakdown, with easy bleeding and bruising. Thrombocytopaenia is typical. Plasma leakage results in haemoconcentration, raising the haemocrit, and loss of plasma proteins into the extracellular space is reflected by the low albumin. The liver is often enlarged and tender, with deranged liver function tests. In severe cases this progresses to circulatory collapse and dengue shock syndrome. Diagnosis is by PCR (polymerase chain reaction) during the initial febrile phase and serology during the later stages. Management is supportive.

Non-falciparum malaria includes disease caused by *Plasmodium vivax* and *ovale*, and to a lesser extent *malariae* and *knowlesi*. *Vivax* is common in Thailand and typically causes less severe disease. Most malaria rapid diagnostic tests are antigen assays that detect only falciparum. Microscopy is the only way to confirm or exclude the diagnosis.

Lassa fever is a viral haemorrhagic fever endemic to West Africa.

Meningococcal septicaemia is a possibility, although one would expect him to be more unwell after five days of disease.

HIV seroconversion illness does not commonly cause bleeding manifestations.

Discuss (6) Improve

Next question

Dengue fever

Dengue fever is a viral infection that can progress to viral haemorrhagic fever (other examples include yellow fever, Lassa fever, Ebola).

Aetiology
- dengue virus is a RNA virus of the genus Flavivirus
- transmitted by the *Aedes aegypti* mosquito
- incubation period of 7 days

Clinical features

Patients with dengue fever can be classified as follows:
- dengue fever:
  - without warning signs
  - with warning signs
- severe dengue (dengue haemorrhagic fever)

Dengue fever
- fever
- headache (often retro-orbital)
- myalgia, bone pain and arthralgia ('break-bone fever')
- pleuritic pain
- facial flushing (dengue)
- maculopapular rash
- haemorrhagic manifestations e.g. positive tourniquet test, petechiae, purpura/ecchymosis, epistaxis
- 'warning signs' include:
  - abdominal pain
  - hepatomegaly
  - persistent vomiting

- clinical fluid accumulation (ascites, pleural effusion)

Severe dengue (dengue haemorrhagic fever)

- this is a form of disseminated intravascular coagulation (DIC) resulting in:
  - thrombocytopenia
  - spontaneous bleeding
- around 20-30% of these patients go on to develop dengue shock syndrome (DSS)

Investigations

- typically blood results
  - leukopenia, thrombocytopenia, raised aminotransferases
- diagnostic tests
  - serology
  - nucleic acid amplification tests for viral RNA
  - NS1 antigen test

Treatment

- entirely symptomatic e.g. fluid resuscitation, blood transfusion etc
- no antivirals are currently available



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians

8 14

2011 Dengue fever review

Postgraduate Medical Journal

Review of dengue fever

6 9

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A 45-year-old man is urgently referred to dermatology clinic by his general practitioner after presenting with erythema migrans on his left calf. The patient was very concerned about the possibility that he had contracted Lyme disease following his recent walking holiday in the Scottish highlands.

The patient reported returning from his holiday 2 weeks ago and first noticed the rash within the last 24 hours. The patient reported that he had undertaken long walks in the countryside wearing shorts but that he had not noticed any ticks becoming attached to his body during his holiday. Aside from the rash on his leg (described below) the patient reported no other symptoms at the present time or during recent weeks. In particular, the patient denied any symptoms involving the central or peripheral nervous systems.

The patient was in generally good physical health and his only past medical history was hypertension treated with ramipril by his GP. The patient had no known allergies to any medications.

Examination of the patient's left leg demonstrated a circular erythematous rash 15 cm in diameter. The rash demonstrated an appearance typical for erythema migrans, featuring a central spot surrounded by a ring of clear skin and an outer ring of erythema. The patient reported that the area covered by the rash had expanded significantly in the 24 hours since he had first noticed it. A detailed examination of the remaining surface of the patient's skin did not identify any other significant rashes. Examination of the peripheral and central nervous system did not demonstrate any signs of focal neurology. Examination of the cardiovascular, respiratory and abdominal systems was unremarkable. There was no evidence of palpable lymphadenopathy.

What is the appropriate management of this patient's erythema migrans?

- Serological testing for pathogenic *Borrelia* species
- Treat with doxycycline for 14 days
- No further investigation or treatment required due to absence of history of tick bite
- Review patient in two weeks and arrange serological tests if onset of symptoms or signs of Lyme borreliosis
- Review patient in two weeks and treat with doxycycline if onset of symptoms or signs of Lyme borreliosis

Submit answer

Reference ranges

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What is the appropriate management of this patient's erythema migrans?

Serological testing for pathogenic *Borrelia* species	5%
Treat with doxycycline for 14 days	85%
No further investigation or treatment required due to absence of history of tick bite	2%
Review patient in two weeks and arrange serological tests if onset of symptoms or signs of Lyme borreliosis	2%
Review patient in two weeks and treat with doxycycline if onset of symptoms or signs of Lyme borreliosis	6%

First line treatment for early Lyme disease is a 14-21 day course of oral doxycycline

Important for me Less important

Lyme borreliosis is the commonest tick-borne infection in the northern hemisphere. It is relatively rare in the UK, with risk being greatest in rural forested areas such as the Scottish highlands, New Forest and Lake District. The illness is caused by pathogenic *Borrelia* species, members of the spirochete phylum.

Clinical features of Lyme borreliosis include erythema migrans in 90% of symptomatic individuals, which tends to occur to 2-40 days following exposure. Erythema migrans has the classical appearance described in the question but may be homogeneous in the early phase and can be confused for cellulitis or a simple insect bite. Rarely, a non-specific febrile illness without rash can occur. Neurological involvement consisting of meningitis or cranial nerve palsy can occur in early infection.

Erythema migrans is a clinical diagnosis and does not require serological confirmation. Therefore, the patient should be treated immediately with a 2-3 week course of oral doxycycline or amoxicillin, even in the absence of a history of tick attachment. Early treatment of erythema migrans is associated with a good clinical outcome and it would be inappropriate to delay treatment until the onset of other symptoms.

Serological testing should be reserved for individuals in which there is diagnostic uncertainty, neurological symptoms or those who are immunocompromised. Individuals presenting following a tick bite should not be treated or undergo investigation in the absence of erythema migrans or other symptoms of Lyme borreliosis.

Duncan C, Carle G, Seaton R. Tick bite and early Lyme borreliosis. *BMJ* 2012;344:e3124.

Discuss (5) Improve

Next question >

Lyme disease

Lyme disease is caused by the spirochaete *Borrelia burgdorferi* and is spread by ticks.

Early features (within 30 days)

- erythema migrans
  - 'bulls-eye' rash is typically at the site of the tick bite
  - typically develops 1-4 weeks after the initial bite but may present sooner
  - usually painless, more than 5 cm in diameter and slowlly increases in size

    - present in around 80% of patients.
- systemic features
    - headache
    - lethargy
    - fever
    - arthralgia





Later features (after 30 days)

- cardiovascular
    - heart block
    - peri/myocarditis
- neurological
    - facial nerve palsy
    - radicular pain
    - meningitis



Investigation

- NICE recommend that Lyme disease can be diagnosed clinically if erythema migrans is present
    - erythema migrans is therefore an indication to start antibiotics
- enzyme-linked immunosorbent assay (ELISA) antibodies to *Borrelia burgdorferi* are the first-line test
    - if negative and Lyme disease is still suspected in people tested within 4 weeks from symptom onset, repeat the ELISA 4-6 weeks after the first ELISA test. If still suspected in people who have had symptoms for 12 weeks or more then an immunoblot test should be done
    - if positive or equivocal then an immunoblot test for Lyme disease should be done

Management of asymptomatic tick bites

- tick bites can be a relatively common presentation to GP practices, and can cause significant anxiety
- if the tick is still present, the best way to remove it is using fine-tipped tweezers, grasping the tick as close to the skin as possible and pulling upwards firmly. The area should be washed

following.

- NICE guidance does not recommend routine antibiotic treatment to patients who've suffered a tick bite

Management of suspected/confirmed Lyme disease

- doxycycline if early disease. Amoxicillin is an alternative if doxycycline is contraindicated (e.g. pregnancy)
  - people with erythema migrans should be commenced on antibiotic without the need for further tests
- ceftriaxone if disseminated disease
- Jarisch-Herxheimer reaction is sometimes seen after initiating therapy: fever, rash, tachycardia after first dose of antibiotic (more commonly seen in syphilis, another spirochaetal disease)



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 11 13

Lyme disease: management

NICE 11 11

2018 Lyme disease guidelines

Dermnet NZ 12 5

Lyme disease

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Media



Lyme disease

Osmosis - YouTube 4 1

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An 80-year-old man presents to the Emergency Department with a 3-day history of headache, photophobia and vomiting. He is pyrexial and tachycardic on arrival. He has a past medical history of hypertension, type 2 diabetes and ischaemic heart disease. He was born in the UK and has never lived outside the UK. On examination, he has neck stiffness and photophobia and is mildly confused.

A lumbar puncture is carried out immediately after the administration of antibiotics and shows results as below. Blood glucose at the same time is 7.8 mmol/L.

Red blood cells	5	(0)
White cells	150	(<3)
Cell count differential	90% lymphocytes	(N/A)
Protein	1.5 g/L	(0.18 - 0.45)
Glucose	2.6 mmol/L	(Relative to blood glucose)

What is the most likely causative organism?

- *Haemophilus influenzae*
- *Listeria monocytogenes*
- *Mycobacterium tuberculosis*
- *Neisseria meningitidis*
- Herpes simplex virus

Submit answer

Reference ranges

Score: **0%**

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Protein	1.5 g/L	(0.18 - 0.45)
Glucose	2.6 mmol/L	(Relative to blood glucose)

What is the most likely causative organism?

Haemophilus influenzae	4%
Listeria monocytogenes	45%
Mycobacterium tuberculosis	24%
Neisseria meningitidis	5%
Herpes simplex virus	22%

Listeria may be suggested by CSF showing a lymphocytic pleocytosis, raised protein and reduced glucose

Important for me Less important

This patient clearly has clinical meningitis, as evidenced by his headache, neck stiffness, photophobia and fever. A degree of confusion suggests that there may also be some encephalitis. The correct answer is ***Listeria monocytogenes***. This diagnosis is suggested by the CSF results showing predominantly lymphocytes, with reduced glucose. Listeria meningitis tends to occur in neonates, pregnant women, the elderly and the immunocompromised. It most commonly presents

with sepsis and meningism and is more likely than other bacterial causes of meningitis to also cause encephalitis or focal neurological deficits.

Haemophilus influenzae is incorrect. This organism can cause meningitis but it mainly does so in infants and children. The CSF would be expected to contain predominantly neutrophils rather than lymphocytes.

Herpes simplex virus is incorrect. HSV might explain why the patient is encephalitic as well as meningitic and you would expect to see a lymphocytosis in the CSF, but the CSF glucose would be normal not low.

Mycobacterium tuberculosis is incorrect. Tuberculous (TB) meningitis could give a similar CSF picture as in this patient, but is less likely given that the patient has no epidemiological risk factors for TB acquisition. TB meningitis also tends to present less acutely.

Neisseria meningitidis is incorrect. In this case, you would expect to see a predominance of neutrophils in the CSF. Lymphocytes can sometimes also be present if there has been a delay between starting antibiotics and performing the lumbar puncture (partially treated meningitis) but this is not the case here where we are told that the lumbar puncture has been done immediately after giving antibiotics.

Discuss (6) | Improve

Next question >

Listeria

Listeria monocytogenes is a Gram-positive bacillus which has the unusual ability to multiply at low temperatures. It is typically spread via contaminated food, typically unpasteurised dairy products. Infection is particularly dangerous to the unborn child where it can lead to miscarriage.

Risk factors

- elderly
- neonates
- immunosuppression
  - especially glucocorticoids
- pregnancy

Features - can present in a variety of ways

- gastroenteritis
  - diarrhoea
- bacteraemia
  - flu-like illness

- central nervous system infection
  - meningoencephalitis
  - ataxia
  - seizures

Investigation
- blood cultures
  - 'tumbling motility' on wet mounts
- cerebrospinal fluid findings:
  - pleocytosis, often lymphocytes (nontuberculous bacteria usually cause a rise in neutrophils)
  - raised protein
  - reduced glucose

Management
- Listeria is sensitive to amoxicillin/ampicillin (cephalosporins usually inadequate)
- Listeria meningitis should be treated with IV amoxicillin/ampicillin and gentamicin

In pregnant women
- pregnant women are almost 20 times more likely to develop listeriosis compared with the rest of the population due to changes in the immune system
- fetal/neonatal infection can occur both transplacentally and vertically during childbirth
- complications include miscarriage, premature labour, stillbirth and chorioamnionitis
- diagnosis can only be made from blood cultures
- treatment is with amoxicillin

AI + Q 123

Next question ›

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Textbooks

High-yield textbook

Extended textbook

Media



Tumbling motility of listeria

Individual - YouTube 22 3

Report broken media

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A 23-year-old man presents to the emergency department with a fever and shortness of breath. He has a past medical history of type 1 diabetes and has had a recent hospital admission with diabetic ketoacidosis. He now has an insulin pump. He does not smoke or drink alcohol.

Observations:

- Heart rate 120 beats per minute
- Blood pressure 92/48 mmHg
- Respiratory rate 22/minute
- Oxygen saturations 94% on room air
- Temperature 38.4C

On examination, he appears diaphoretic and unwell. Chest auscultation reveals coarse crackles at the right base.

Blood tests:

Hb	138 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	14.7 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	8.2 mmol/L	(2.0 - 7.0)
Creatinine	99 µmol/L	(55 - 120)
CRP	221 mg/L	(< 5)

A chest -ray demonstrates consolidation in the right lower zone.

He is commenced on empirical broad-spectrum antibiotics to treat sepsis secondary to pneumonia.

At 24 hours, a blood culture grows *Klebsiella pneumoniae* , which is identified as producing carbapenemase.

What is the most appropriate antibiotic choice?

- Ceftriaxone
- Co-amoxiclav
- Meropenem
- Piperacillin/tazobactam
- Tigecycline

Submit answer

Reference ranges

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- Oxygen saturations 94% on room air
- Temperature 38.4C

On examination, he appears diaphoretic and unwell. Chest auscultation reveals coarse crackles at the right base.

Blood tests:

Hb	138 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	14.7 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	8.2 mmol/L	(2.0 - 7.0)
Creatinine	99 µmol/L	(55 - 120)
CRP	221 mg/L	(< 5)

A chest -ray demonstrates consolidation in the right lower zone.

He is commenced on empirical broad-spectrum antibiotics to treat sepsis secondary to pneumonia.

At 24 hours, a blood culture grows *Klebsiella pneumoniae* , which is identified as producing carbapenemase.

What is the most appropriate antibiotic choice?

Ceftriaxone	8%
Co-amoxiclav	5%
Meropenem	29%
Piperacillin/tazobactam	27%
Tigecycline	**32%**

Carbapenemase producing enterobacteriaceae (CPE) are multi-drug resistant coliforms. They can be treated with polymyxins (e.g. colistin), tigecycline, fosfomycin or aminoglycosides (e.g. gentamicin)

Important for me Less important

Tigecycline is the correct answer. The patient has developed pneumonia and sepsis secondary to infection with *Klebsiella pneumoniae* , which has been identified as a carbapenemase-producing organism. This is a class of bacteria known as carbapenemase-producing enterobacteriaceae. These are gram-negative bacteria that are resistant to carbapenem and extended-spectrum cephalosporin antibiotics. Effective antibiotics against this class of bacteria include polymyxins (e.g. colistin), tigecycline, fosfomycin or aminoglycosides (e.g. gentamicin). Risk factors for CPE infection include immunosuppression (e.g. diabetes) and previous hospital admission.

Meropenem is incorrect. This bacteria is carbapenem-resistant. Meropenem is a carbapenem.

Ceftriaxone is incorrect. CPE are resistant to the cephalosporin class of medication. This is effective against a wide variety of gram-negative and gram-positive aerobes and anaerobes and would be a reasonable choice for the treatment of hospital-acquired pneumonia on an empirical basis. However, it is not effective against CPE.

Piperacillin/tazobactam is incorrect. This class of antibiotic is effective against a broad spectrum of gram-positive and negative bacteria including *Pseudomonas* but is not effective against CPE.

Co-amoxiclav is incorrect. This has a wide spectrum of activity against various gram-negative and gram-positive aerobes but is not effective against CPE.

Discuss (6) Improve

Next question

Carbapenems

Extended Spectrum Beta-Lactamases (ESBL) are enzymes produced by certain bacteria that have developed the ability to break down and resist the effects of a wide range of beta-lactam antibiotics. These antibiotics include penicillins and cephalosporins. *Escherichia coli* and *Klebsiella* are the most common ESBL-producing bacteria found in clinical practice.

Carbapenems are a class of β-lactam antibiotics that are resistant to most β-lactamases. They are therefore particularly useful in the treatment of infections caused by ESBL-producing bacteria.

Examples

- imipenem: usually combined with cilastatin, a renal dehydropeptidase I inhibitor to decrease the renal degradation of imipenem
- meropenem

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **24%**

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A 31-year-old gentleman presents with fever, headache, abdominal pain and a rash on the chest 3 weeks after visiting South America.

On examination the temperature is 38.2ºC. There is a rash on the chest consisting of rose-coloured blanching papules. The respiratory rate is 20 breaths/min and the heart rate is 58 beats per minute. The chest is clear to auscultation. The abdomen is diffusely tender and there is mild splenomegaly.

Initial blood results are as follows:

Hb	128 g/l
Platelets	184 * 10^9/l
WBC	3.9 * 10^9/l
Na^+	131 mmol/l
K^+	3.3 mmol/l
Urea	7.2 mmol/l
Creatinine	141 µmol/l
Bilirubin	46 µmol/l
ALP	147 u/l
ALT	96 u/l
Albumin	38 g/l
CRP	52 mg/l

What is the most appropriate initial antimicrobial therapy?

- Ampicillin
- Chloramphenicol
- Trimethoprim-sulfamethoxazole
- Cefotaxime
- Streptomycin

Submit answer

Reference ranges

Score: **0%**

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ALP	147 u/l
ALT	96 u/l
Albumin	38 g/l
CRP	52 mg/l

What is the most appropriate initial antimicrobial therapy?

Ampicillin	13%
Chloramphenicol	15%
Trimethoprim-sulfamethoxazole	21%
Cefotaxime	40%
Streptomycin	12%

This is a fairly classical presentation of typhoid fever, also known as enteric fever. This is a potentially fatal multisystemic illness caused primarily by *Salmonella enterica*.

The rash here refers to rose spots which occur in up to 30% of people infected with this organism. Characteristically, rose spots are seen in untreated typhoid fever. They usually occur between the second and fourth week of the illness. They characteristically present as groups of 5-15 pink blanching papules distributed between the level of the nipples and umbilicus.

The treatment of choice is cefotaxime or ceftriaxone.

Ciprofloxacin may be used as an alternative in sensitive organisms.

Discuss (10) | Improve

Next question >

Enteric fever (typhoid/paratyphoid)

The *Salmonella* group contains many members, most of which cause diarrhoeal diseases. They are aerobic, Gram-negative rods which are not normally present as commensals in the gut.

Typhoid and paratyphoid are caused by *Salmonella typhi* and *Salmonella paratyphi* (types A, B & C) respectively. They are often termed enteric fevers, producing systemic symptoms such as headache, fever, arthralgia.

Pathophysiology

- typhoid is transmitted via the faecal-oral route (also in contaminated food and water)

Features

- initially systemic upset as above
- relative bradycardia
- abdominal pain, distension
- constipation: although *Salmonella* is a recognised cause of diarrhoea, constipation is more common in typhoid
- rose spots: present on the trunk in 40% of patients, and are more common in paratyphoid

Possible complications include

- osteomyelitis (especially in sickle cell disease where *Salmonella* is one of the most common pathogens)
- GI bleed/perforation

- meningitis
- cholecystitis
- chronic carriage (1%, more likely if adult females)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

6 9

Typhoid fever review

Suggest link

Report broken link

Media



Salmonella - a quick introduction and overview

Let's Learn About Bugs - YouTube 4 0



Salmonella

ATP - YouTube 7 1



Salmonella

SketchyMedical - YouTube 6 3

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Score: **24%**

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A 48-year-old farmer presents with a four-day history of headache, pyrexia and vomiting. He has recently been undergoing chemotherapy for chemotherapy for mantle cell lymphoma and successfully completed his fourth cycle three days ago. During his treatment, he has tried to maintain an active lifestyle and has continued to work on his dairy farm. He has no other past medical history, he does not smoke or drink. On examination, the patient is drowsy and pyrexic at 38.6 degrees. You note no rashes on his skin. His displays neck stiffness and photophobia. You are unable to formally perform a neurological examination but you note no obvious facial asymmetry and the patient is moving all four limbs. Both plantars are downgoing.

Blood tests are as follows:

Hb	98 g/l
Platelets	78 * 10^9/l
WBC	0.9 * 10^9/l
Neutrophils	0.3 * 10^9/l

Na^+	146 mmol/l
K^+	4.3 mmol/l
Urea	8 mmol/l
Creatinine	99 µmol/l
CRP	170 mg/l

A lumbar puncture is performed and the cerebrospinal fluid examination is as follows:

WCC	200 x 10^6/litre (70% neutrophil 25% lymphocytes)
RBC	4 x 10^6/litre
Glucose	1.7 mmol/l (normal 3.3-4.4 mmol/l)
Microscopy	No organisms on gram stain
Appearance	cloudy

The patient is immediately commenced on intravenous ceftriaxone for suspected bacterial meningitis. At 48 hours after initial admission, blood and cerebrospinal fluid cultures are still awaited. The patient has demonstrated no change in clinical state. What is an appropriate additional therapy?

- Hourly neurological monitoring

- Intravenous acyclovir
- Intravenous ampicillin
- Intravenous rifampicin
- Intravenous amphotericin B

Submit answer

Reference ranges

Score: **0%**

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Appearance	cloudy

The patient is immediately commenced on intravenous ceftriaxone for suspected bacterial meningitis. At 48 hours after initial admission, blood and cerebrospinal fluid cultures are still awaited. The patient has demonstrated no change in clinical state. What is an appropriate additional therapy?

Hourly neurological monitoring 2%

Intravenous acyclovir	19%
Intravenous ampicillin	34%
Intravenous rifampicin	11%
Intravenous amphotericin B	33%

A diagnosis of meningitis is clear. However, the context is that of a patient who is immunocompromised with recent chemotherapy, presenting with a relatively subacute cause of neutropenic sepsis. The additional information that he continues to work on a dairy with possible exposure to unpasteurised milk increases the chance of listeria exposure, for which intravenous ampicillin is the most appropriate treatment. The patient has demonstrated no improvement in 48 hours; antibiotic escalation would be appropriate. It should be noted that on CSF examination, the absence of an organism on Gram staining does not equate to the absence of an organism! Only one-third of listeria meningitis patients are likely to grow an organism on Gram stain. In addition, listeria meningitis is a classic example of bacterial meningitis when a significant proportion of lymphocytes may be in CSF (>25%), instead of predominant neutrophilia. These include TB, rickettsial and cryptococcal meningitis. With no mention of previous or active TB exposure and no pulmonary symptoms on a relatively short history, TB meningitis is only a low-risk possibility. Intravenous amphotericin B would be appropriate for fungal meningitis only.

Discuss (9) Improve

Next question

Listeria

Listeria monocytogenes is a Gram-positive bacillus which has the unusual ability to multiply at low temperatures. It is typically spread via contaminated food, typically unpasteurised dairy products. Infection is particularly dangerous to the unborn child where it can lead to miscarriage.

Risk factors

- elderly
- neonates
- immunosuppression
  - especially glucocorticoids
- pregnancy

Features - can present in a variety of ways

- gastroenteritis
  - diarrhoea
- bacteraemia
  - flu-like illness
- central nervous system infection
  - meningoencephalitis
  - ataxia
  - seizures

Investigation
- blood cultures
  - 'tumbling motility' on wet mounts
- cerebrospinal fluid findings:
  - pleocytosis, often lymphocytes (nontuberculous bacteria usually cause a rise in neutrophils)
  - raised protein
  - reduced glucose

Management
- Listeria is sensitive to amoxicillin/ampicillin (cephalosporins usually inadequate)
- Listeria meningitis should be treated with IV amoxicillin/ampicillin and gentamicin

In pregnant women
- pregnant women are almost 20 times more likely to develop listeriosis compared with the rest of the population due to changes in the immune system
- fetal/neonatal infection can occur both transplacentally and vertically during childbirth
- complications include miscarriage, premature labour, stillbirth and chorioamnionitis
- diagnosis can only be made from blood cultures
- treatment is with amoxicillin

AI + Q 123

Next question ›

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Textbooks

High-yield textbook

Extended textbook

Media



Tumbling motility of listeria

Individual - YouTube 22 3

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Jenna is an 18-year-old woman who was initially admitted with a fever and disseminated rash. She had not been previously vaccinated and was in contact with her 2-year-old cousin who had developed a fever and disseminated blisters and vesicles containing clear fluid. She began to have a fever and flu-like symptoms 2 weeks after seeing her cousin. Similar clear-fluid filled vesicles and blisters developed 3 days later and she was admitted for further observation.

After 3 days, she noticed that while most of her skin lesions are healing, one of the lesions on the thigh appears to be red and becoming hot to touch. An area of skin approximately 3x3cm was erythematous. The skin was marked and she was commenced on IV flucloxacillin. Over the coming 12 hours, the erythema around this lesion continued to spread. The pain around her leg increased in intensity, requiring morphine to take the edge off the pain. A blueish discolouration begins to develop around the rash.

Given the likely complication that has developed, what is the likely organism that has caused the complication?

- *Enterococcus faecalis*
- *Clostridium perfringens*
- *Staphylococcus aureus*
- *Streptococcus bovis*
- β- haemolytic Group A *Streptococcus*

Submit answer

Reference ranges

Score: **0%**

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Given the likely complication that has developed, what is the likely organism that has caused the complication?

Enterococcus faecalis	2%
Clostridium perfringens	22%
Staphylococcus aureus	13%
Streptococcus bovis	1%
β- haemolytic Group A *Streptococcus*	62%

Chickenpox is a risk factor for invasive group A streptococcal soft tissue infections including necrotizing fasciitis

Important for me Less important

The development of fevers along with blisters and vesicles raises the suspicion that this is caused by chickenpox. Besides, this is more likely as she has been exposed to a child who has had similar symptoms. Chickenpox tends to be milder in children but can cause significant morbidity in adults.

The development of a rapidly evolving rash along with significant pains out of proportion to the rash seen should always raise alarm bells for necrotising fasciitis. Blueish discolouration of the skin is also suggestive of this. A surgical review should be sought immediately.

Invasive group A *Streptococcus*, a β-haemolytic *Streptococcus*, has been implicated as the cause for necrotizing fasciitis in patients with chickenpox. Extremely broad-spectrum antibiotics are initially used with the choices tailored to bacterial sensitivities when known.

Staphylococcus aureus can also result in necrotising fasciitis but is more commonly associated with patients who have other underlying medical conditions like diabetes.

Enterococcus faecalis does not tend to cause skin infections and are often associated with infections like endocarditis.

Streptococcus bovis is a gamma-haemolytic *Streptococcus* is most often associated with colorectal cancer associated endocarditis. It is not associated with skin infections.

Clostridium perfringens can cause necrotising fasciitis and presents as gas gangrene. This will present with crepitus under the skin which is not seen here.

Discuss (2) Improve

Next question >

Chickenpox ★

Chickenpox is caused by primary infection with varicella zoster virus. Shingles is a reactivation of the dormant virus in dorsal root ganglion

Chickenpox is highly infectious

- spread via the respiratory route
- can be caught from someone with shingles
- infectivity = 4 days before rash, until 5 days after the rash first appeared*
- incubation period = 10-21 days

Clinical features (tend to be more severe in older children/adults)

- fever initially
- itchy, rash starting on head/trunk before spreading. Initially macular then papular then vesicular
- systemic upset is usually mild

Management is supportive

- keep cool, trim nails
- calamine lotion

- school exclusion: NICE state the following: *Advise that the most infectious period is 1-2 days before the rash appears, but infectivity continues until all the lesions are dry and have crusted over (usually about 5 days after the onset of the rash).*
- immunocompromised patients and newborns with peripartum exposure should receive varicella zoster immunoglobulin (VZIG). If chickenpox develops then IV aciclovir should be considered

A common complication is secondary bacterial infection of the lesions

- NSAIDs may increase this risk
- whilst this commonly may manifest as a single infected lesion/small area of cellulitis, in a small number of patients invasive group A streptococcal soft tissue infections may occur resulting in necrotizing fasciitis

Rare complications include

- pneumonia
- encephalitis (cerebellar involvement may be seen)
- disseminated haemorrhagic chickenpox
- arthritis, nephritis and pancreatitis may very rarely be seen



© Image used on license from Radiopaedia

Chest x-ray showing miliary opacities secondary to healed varicella pneumonia. Multiple tiny calcific miliary opacities noted throughout both lungs. These are of uniform size and dense suggesting calcification. There is no focal lung parenchymal mass or cavitating lesion seen. The appearances are characteristic for healed varicella pneumonia.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Greenbook 8 6

Varicella

Public Health England 9 6

Chapter 9: managing specific infectious diseases

Suggest link Report broken link

Media



Varicella zoster virus

Osmosis - YouTube 6 0

Report broken media

Score: **24%**

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A 44-year-old vet presents with a fever to his GP. He has had the fever for 4 days and it seems to come and go. He also mentions that he has felt generally lethargic for the past week, with occasional lower back pain and aching limbs. On examination, he is tender in his sacral region and has an enlarged liver and spleen. Blood tests reveal the following:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	160 * 10^9/L	(150 - 400)
WBC	3.8 * 10^9/L	(4.0 - 11.0)
Bilirubin	7 µmol/L	(3 - 17)
ALP	50 u/L	(30 - 100)
ALT	23 u/L	(3 - 40)
Î³GT	10 u/L	(8 - 60)
Albumin	43 g/L	(35 - 50)

Which of the following pathogens is the most likely cause of this man's presentation?

- Cryptosporidium
- Brucella
- Bartonella henselae
- Echinococcus granulosis
- Leptospira

Submit answer

Reference ranges

Score: **0%**

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ALT	23 u/L	(3 - 40)
Î³GT	10 u/L	(8 - 60)
Albumin	43 g/L	(35 - 50)

Which of the following pathogens is the most likely cause of this man's presentation?

Cryptosporidium	2%
Brucella	64%
Bartonella henselae	16%
Echinococcus granulosis	7%
Leptospira	11%

Unwell vet with fever, malaise, arthralgia and lower back pain ? Brucellosis

Important for me Less important

This man presents with features suggestive of brucellosis, including fluctuating fevers, malaise, arthralgia, sacroiliitis, hepatosplenomegaly and mild leukopenia. Brucellosis is a zoonosis: an infectious disease that jumps from a non-human animal to humans. It is caused by the gram-negative, rod-shaped *Brucella* species of bacteria. It is more common in the Middle East and in

those that work with animals, such as this man. It is spread by aerosol and considered a bioterrorism agent. Rose Bengal plate test is used in suspected Brucellosis. One can remember this as B-Rose in B-Ruce-losis.

Cryptosporidiosis is a protozoal disease that typically affects the immunocompromised and causes diarrhoea.

Bartonella henselae is a bacteria that causes cat-scratch disease, another disease that is more common in vets. Although it may present with flu-like symptoms, it does not typically cause hepatosplenomegaly. There is typically a history of a bite or scratch from a cat, with a lesion developing over the area and painful lymphadenopathy. As a side note *Bartonella bacilliformis* can cause Oroya fever, which may present with hepatosplenomegaly and fever, but typically there is lymphadenopathy also, with marked haemolysis.

Echinococcus granulosis is a tapeworm that causes hydatid disease, another disease that is more common in vets. However, hydatid disease causes cysts that typically affect the liver or lungs, and is more likely to present with abdominal pain, jaundice, weight loss, and if affecting the lungs then a cough and shortness of breath.

Leptospirosis is a possible diagnosis here, as it often presents in vets, and can present with flu-like illness and hepatosplenomegaly. The sacral tenderness, leukopenia and normal LFTs make this less likely. Leptospirosis will typically cause leukocytosis, anaemia, thrombocytopenia, even in the absence of DIC. Even when clinical manifestations are mild, hyperbilirubinemia is often present and mild elevations of serum transaminases are frequently seen. In exams, leptospirosis will normally present with bilateral conjunctival suffusion, which is conjunctivitis without exudate. However, in reality, this occurs in <60% of patients.

Discuss (3) | Improve

Next question >

Brucellosis

Brucellosis is a zoonosis more common in the Middle East and in farmers, vets and abattoir workers. Four major species cause infection in humans: *B. melitensis* (sheep), *B. abortus* (cattle), *B. canis* and *B. suis* (pigs). Brucellosis has an incubation period 2 - 6 weeks.

Features

- non-specific: fever, malaise
- hepatosplenomegaly
- sacroiliitis: spinal tenderness may be seen
- complications: osteomyelitis, infective endocarditis, meningoencephalitis, orchitis
- leukopenia often seen

Diagnosis

- the Rose Bengal plate test can be used for screening but other tests are required to confirm the diagnosis
- Brucella serology is the best test for diagnosis
- blood and bone marrow cultures may be suitable in certain patients, but these tests are often negative

Management

- doxycycline and streptomycin

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

WHO

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Brucellosis guidelines

Suggest link Report broken link

Score: **24%**

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A 45-year-old man is reviewed in the HIV clinic. He was diagnosed with HIV 25 years ago and has enjoyed good health on highly active antiretroviral treatment until around 4 years ago when he had an episode of *Pneumocystis jirovecii* pneumonia.

Today he is somewhat confused. His partner reports that he has been complaining of headaches for several weeks and has also been uncharacteristically aggressive.

His latest CD4 count is 29 cells/μl. An MRI (T1 C+) is requested:



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What is the most appropriate treatment?

- Sulfadiazine + pyrimethamine
- Sulfadiazine + pyrimethamine + steroids
- Steroids + methotrexate
- Rifampicin + isoniazid + pyrazinamide + ethambutol + steroids
- Amphotericin B

Submit answer

Reference ranges

Score: **0%**

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A 45-year-old man is reviewed in the HIV clinic. He was diagnosed with HIV 25 years ago and has enjoyed good health on highly active antiretroviral treatment until around 4 years ago when he had an episode of *Pneumocystis jirovecii* pneumonia.

Today he is somewhat confused. His partner reports that he has been complaining of headaches for several weeks and has also been uncharacteristically aggressive.

His latest CD4 count is 29 cells/µl. An MRI (T1 C+) is requested:



© Image used on license from Radiopaedia

What is the most appropriate treatment?

Sulfadiazine + pyrimethamine	16%
Sulfadiazine + pyrimethamine + steroids	36%
Steroids + methotrexate	35%
Rifampicin + isoniazid + pyrazinamide + ethambutol + steroids	8%
Amphotericin B	5%

HIV, neuro symptoms, single brain lesions with homogenous enhancement - CNS lymphoma

Important for me Less important

The MRI scan demonstrates a large multilobulated mass in the right frontal lobe. It homogeneously enhances and extends to involve the caudate and the periventricular area. There is also a significant mass effect. These findings in addition to the history are highly suggestive of primary CNS lymphoma. The most appropriate initial treatment is steroids (which should reduce tumour size and hence mass effect) and methotrexate. Other future options include whole brain irradiation and surgery.

You would not expect the enhancement to be homogenous with toxoplasmosis, it is usually in a ring pattern.

Discuss (5) Improve

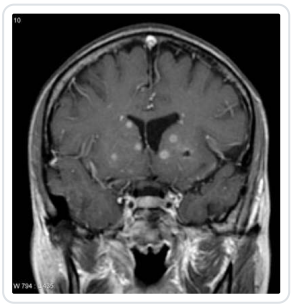
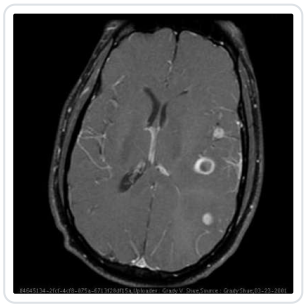
Next question

HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

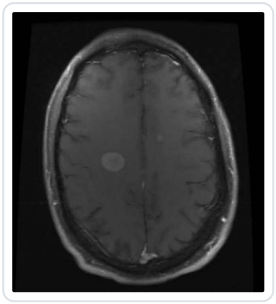




Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions

- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis
- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis
- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus
- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema

- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination
- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link Report broken link

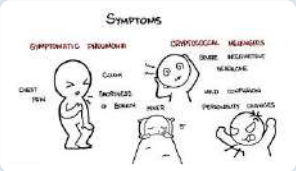
Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

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Report broken media

Score: **24%**

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A 34-year-old man presents to the emergency department with two weeks of fevers, malaise, myalgia, arthralgia and low back pain. He has no significant past medical history. He does not take any medications. He works as a veterinarian. There is no recent travel.

Observations:

- Heart rate 94 beats per minute
- Blood pressure 121/88 mmHg
- Respiratory rate 19/minute
- Oxygen saturation 96% on room air
- Temperature 38.2ºC

On examination, there is focal tenderness at the left sacroiliac joint. Cardiovascular, respiratory and abdominal examinations are unremarkable. He is diaphoretic and an associated foul smell is noted. There is no joint swelling.

Urinalysis is unremarkable.

Plain radiography of the chest is normal.

Blood tests:

Hb	138 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	3.4 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	45 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	89 u/L	(30 - 100)
ALT	35 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

What is the likely diagnosis?

- Brucellosis
- HIV seroconversion
- Leptospirosis
- Q fever
- Tuberculosis

Submit answer

Reference ranges

Score: **0%**

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ALT	35 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

What is the likely diagnosis?

Brucellosis	74%
HIV seroconversion	2%
Leptospirosis	7%
Q fever	15%
Tuberculosis	2%

Unwell vet with fever, malaise, arthralgia and lower back pain ? Brucellosis

Important for me Less important

Brucellosis is the correct answer. The patient presents with fevers, malaise, arthralgia, myalgia and low back pain. The location of the back pain suggests sacroiliitis. Profuse sweating associated with a foul smell is typical of this condition. It is often said to be 'hay-like'. His work as a veterinarian is a relevant occupational exposure.

Leptospirosis is incorrect. This can cause a febrile illness with arthralgia and myalgia. However, it is typically associated with subconjunctival haemorrhages and headache and pulmonary haemorrhage and jaundice in severe cases. Foul-smelling sweat is not a typical feature.

Q fever is incorrect. While he works as a vet and this is a relevant occupational exposure, this condition is typically characterised by an atypical pneumonia, which is absent in this case. Sacroiliac involvement is not a feature.

Tuberculosis is incorrect. This can cause arthralgia, fevers and in rare cases can involve the sacroiliac joint. However, the occupational exposure and foul-smelling sweat making brucellosis the more likely diagnosis, especially in the absence of a relevant travel history.

HIV seroconversion is incorrect. While this can cause a febrile illness, myalgia, arthralgia and a low white blood cell count. However, the relevant occupational exposure, sacroiliac joint involvement and foul-smelling sweat make a diagnosis of brucellosis more likely.

Discuss (2) Improve

Next question

Brucellosis

Brucellosis is a zoonosis more common in the Middle East and in farmers, vets and abattoir workers. Four major species cause infection in humans: *B. melitensis* (sheep), *B. abortus* (cattle), *B. canis* and *B. suis* (pigs). Brucellosis has an incubation period 2 - 6 weeks.

Features

- non-specific: fever, malaise
- hepatosplenomegaly
- sacroiliitis: spinal tenderness may be seen
- complications: osteomyelitis, infective endocarditis, meningoencephalitis, orchitis
- leukopenia often seen

Diagnosis

- the Rose Bengal plate test can be used for screening but other tests are required to confirm the diagnosis
- Brucella serology is the best test for diagnosis
- blood and bone marrow cultures may be suitable in certain patients, but these tests are often negative

Management

- doxycycline and streptomycin



Next question

Textbooks

High-yield textbook

Extended textbook

Links

WHO

2 5

Brucellosis guidelines

Suggest link Report broken link

Score: **24%**

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A 32-year-old man has been brought to the hospital with severe shortness of breath.

Initial observations with the paramedics were taken. These included a respiratory rate of 32/min, oxygen saturations of 72% on room air, a pulse of 120/min, a blood pressure of 123/72mmHg, and a temperature of 38.3ºC.

On examination in the emergency department, he is sat on the bed leaning forwards. There is evidence of drooling and a vomit bowl full of saliva is next to him. There is audible stridor from the edge of the bed. Oxygen has now been applied and saturations are 90% on 15L.

A neck x-ray was performed due to concerns for a foreign body and has shown a 'thumb sign'.

What is the most appropriate next step in management?

- Continuous positive airway pressure (CPAP)
- Endotracheal intubation
- High flow oxygen
- Intramuscular adrenaline
- Nebulised adrenaline

Submit answer

Reference ranges

Score: **0%**

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A neck x-ray was performed due to concerns for a foreign body and has shown a 'thumb sign'.

What is the most appropriate next step in management?

Continuous positive airway pressure (CPAP)	3%
Endotracheal intubation	62%
High flow oxygen	3%
Intramuscular adrenaline	10%
Nebulised adrenaline	22%

Endotracheal intubation may be necessary to protect the airway in acute epiglottitis

Important for me Less important

The above scenario describes a severe case of acute epiglottitis. Patients approaching extremis tend to lean forwards to aid their breathing and may have pooling of saliva due to an inability to swallow. The 'thumb sign' on the neck x-ray is indicative of an oedematous epiglottitis. Given the significant desaturation in oxygen along with audible stridor, the most appropriate intervention would be endotracheal intubation.

Given the degree of obstruction in this case, urgent anaesthetic input should be sourced due to the high likelihood that this patient will require intubation. This should not be delayed to trial CPAP. It is also possible that CPAP will lead to the displacement of the epiglottis and worsen the obstruction.

High flow oxygen can be used in patients that have low oxygen saturation who are otherwise stable. This would not be appropriate in the above scenario given the stridor and a need to urgently secure the airway.

Intramuscular adrenaline would be beneficial if this was stridor associated with anaphylaxis, however, there is no evidence of this from the scenario.

Nebulised adrenaline can be used in croup but is not a part of management in epiglottitis.

Discuss (2) | Improve

Next question >

Acute epiglottitis ★

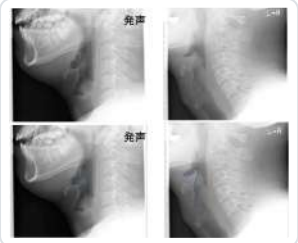
Acute epiglottitis is rare but serious infection caused by *Haemophilus influenzae* type B. Prompt recognition and treatment is essential as airway obstruction may develop. Epiglottitis was generally considered a disease of childhood but in the UK it is now more common in adults due to the immunisation programme. The incidence of epiglottitis has decreased since the introduction of the Hib vaccine.

Features

- rapid onset
- high temperature, generally unwell
- stridor
- drooling of saliva
- 'tripod' position: the patient finds it easier to breathe if they are leaning forward and extending their neck in a seated position

Diagnosis is made by direct visualisation (only by senior/airway trained staff, see below). However, x-rays may be done, particularly if there is concern about a foreign body:

- a lateral view in acute epiglottis will show swelling of the epiglottis - the 'thumb sign'
- in contrast, a posterior-anterior view in croup will show subglottic narrowing, commonly called the 'steeple sign'



Management

- immediate senior involvement, including those able to provide emergency airway support (e.g. anaesthetics, ENT)
  - endotracheal intubation may be necessary to protect the airway
- if suspected do NOT examine the throat due to the risk of acute airway obstruction
  - the diagnosis is made by direct visualisation but this should only be done by senior staff who are able to intubate if necessary
- oxygen
- intravenous antibiotics

AI + Q 123

Next question >

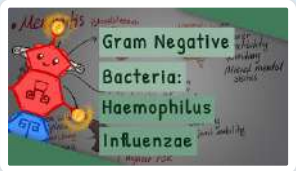
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Textbooks

High-yield textbook

Extended textbook

Media



Haemophilus influenzae

ATP - YouTube

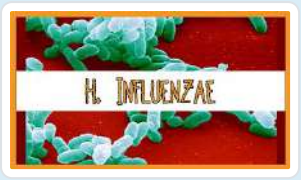
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Stridor in children (causes, inspiratory and expiratory stridor, pathophysiology)

Armando Hasudungan - YouTube

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Haemophilus influenzae

Dirty Medicine - YouTube

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Score: **24%**

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A 25-year-old woman is started on antiretroviral therapy for HIV. She attends the GP 1 week later complaining of dizziness and nightmares. She is unable to work due to feeling muddled and disconnected. Which drug is responsible?

- Abacavir
- Efaverinz
- Lamivudine
- Tenofovir
- Zidovudine

Submit answer

Reference ranges

Score: **0%**

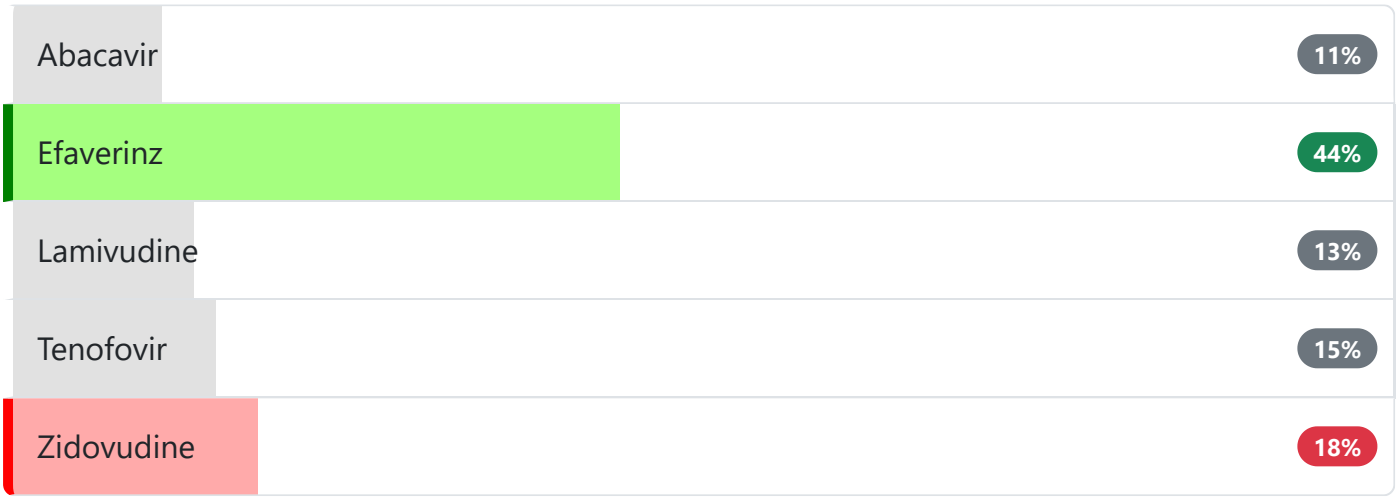
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A 25-year-old woman is started on antiretroviral therapy for HIV. She attends the GP 1 week later complaining of dizziness and nightmares. She is unable to work due to feeling muddled and disconnected. Which drug is responsible?



Efaverinz is a non-nucleoside reverse transcriptase inhibitor (NNRTI) and is frequently known to cause disturbing dreams and other cognitive disturbances in 50% of patients in the first month of treatment.

The most concerning side effect of Abacavir is a hypersensitivity reaction that can lead to fever, malaise, nausea and skin rash which can be very severe. It is strongly associated with a specific allele at the human leukocyte antigen B locus, HLA-B*57:01.

Lamivudine and Zidovudine are nucleoside analogue reverse transcriptase inhibitors (NRTIs). The NRTIs were the first class of antiretroviral drugs developed. As with all drugs in their class, they can cause a potentially fatal lactic acidosis & severe hepatomegaly with steatosis when used alone or in combination with other antiretrovirals.

Tenofovir is a nucleotide analogue reverse transcriptase inhibitors (NtRTI), it is a prodrug, the commonest side effects are gastrointestinal.

Discuss (3) | Improve

Next question

HIV: management

Antiretroviral therapy (ART) involves a combination of at least three drugs, typically two nucleoside reverse transcriptase inhibitors (NRTI) and either a protease inhibitor (PI) or a non-nucleoside reverse transcriptase inhibitor (NNRTI). This combination both decreases viral replication but also reduces the risk of viral resistance emerging

Following the 2015 BHIVA guidelines it is now recommended that patients start ART as soon as they have been diagnosed with HIV, rather than waiting until a particular CD4 count, as was previously advocated.

Entry inhibitors

- maraviroc (binds to CCR5, preventing an interaction with gp41), enfuvirtide (binds to gp41, also known as a 'fusion inhibitor')
- prevent HIV-1 from entering and infecting immune cells

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), abacavir, emtricitabine, didanosine, lamivudine, stavudine, zalcitabine, tenofovir
- general NRTI side-effects: peripheral neuropathy
- tenofovir: used in BHIVAs two recommended regime NRTI. Adverse effects include renal impairment and ostesoporosis
- zidovudine: anaemia, myopathy, black nails
- didanosine: pancreatitis

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz
- side-effects: P450 enzyme interaction (nevirapine induces), rashes

Protease inhibitors (PI)

- examples: indinavir, nelfinavir, ritonavir, saquinavir
- side-effects: diabetes, hyperlipidaemia, buffalo hump, central obesity, P450 enzyme inhibition
- indinavir: renal stones, asymptomatic hyperbilirubinaemia
- ritonavir: a potent inhibitor of the P450 system

Integrase inhibitors

- block the action of integrase, a viral enzyme that inserts the viral genome into the DNA of the host cell
- examples: raltegravir, elvitegravir, dolutegravir



Next question

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

2016 HIV-1 infection guidelines

7 4

University of Liverpool

HIV Drug Interactions Checker

12 6

Suggest link

Report broken link

Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube

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Antiretroviral drugs

Medinaz - YouTube

38 2

Report broken media

Score: **24%**

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A 57-year-old male with chronic hepatitis B infection presents to the Emergency Department with increasing confusion. His wife states that his specialist has not started him on antivirals yet. He was diagnosed six months ago. His past medical history includes tuberculosis that has been successfully treated 10 years ago.

His wife stated also that he slept more than usual in the last week and he was more lethargic. On examination the patient was confused, disoriented with a yellowish discoloration of his eyes. There is moderate peripheral oedema and moderate ascites with no signs of hepatomegaly.

Blood investigations were as follows:

Hb	102 g/l
Platelets	250 * 10^9/l
WBC	9.5 * 10^9/l
Bilirubin	42 µmol/l
ALP	135 u/l
ALT	370 u/l
γGT	70 u/l
Albumin	32 g/l
α-fetoprotein	15 kU/l

What is the most likely cause of this presentation?

- Portal vein thrombosis
- Budd-Chiari syndrome
- Hepatitis D infection
- Hepatocellular carcinoma
- Normal progression of the disease

Submit answer

Reference ranges

Score: **0%**

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Albumin	32 g/l
α-fetoprotein	15 kU/l

What is the most likely cause of this presentation?

Portal vein thrombosis	8%
Budd-Chiari syndrome	9%
Hepatitis D infection	43%
Hepatocellular carcinoma	24%
Normal progression of the disease	16%

AI

This patient has developed acute liver decompensation. Hepatitis D infection is an uncommon but important cause of this presentation. It typically occurs in a patient with a history of stable chronic

hepatitis B infection that suddenly acutely deteriorates.

Budd-Chiari syndrome (BCS) and portal vein thrombosis are not the correct options because it is unlikely to have any of them in the absence of hepatomegaly. In addition, the high level of the transaminase enzyme suggests viral infection rather than thrombus formation. Note that the transaminase enzyme should be only mildly elevated in BCS. Fever and nausea are also important symptoms that are usually present in BCS.

Hepatocellular carcinoma usually presents with very high levels of alpha-fetoprotein.

Discuss (13) Improve

Next question

Hepatitis D

Hepatitis D is a single stranded RNA virus that is transmitted parenterally. It is an incomplete RNA virus that requires hepatitis B surface antigen to complete its replication and transmission cycle.

It is transmitted in a similar fashion to hepatitis B (exchange of bodily fluids) and patients may be infected with hepatitis B and hepatitis D at the same time.

Hepatitis D terminology:

- Co-infection: Hepatitis B and Hepatitis D infection at the same time.
- Superinfection: A hepatitis B surface antigen positive patient subsequently develops a hepatitis D infection.

Superinfection is associated with high risk of fulminant hepatitis, chronic hepatitis status and cirrhosis.

Diagnosis is made via reverse polymerase chain reaction of hepatitis D RNA. Interferon is currently used as treatment, but with a poor evidence base.

AI + Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Media



Hepatitis

Podmedics - YouTube 1 0



Viral hepatitis

Osmosis - YouTube 0 0

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Score: **24%**

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A 32-year-old woman attends the infectious diseases clinic for an urgent review. The patient had presented to the emergency department the previous weekend complaining of various constitutional symptoms. A set of blood cultures had been taken as the patient was febrile at presentation. Assessment in the emergency department had not identified a clear cause for the patient's illness and she had been discharged home with advice to attend her GP surgery if her symptoms did not resolve in the coming days. The patient had been recalled to hospital for review after the blood cultures taken during her presentation yielded a positive result. Selected investigation results from the patient's emergency department attendance are given below.

At her review appointment, the patient recounted her recent symptoms. She described a 3-week history of intermittent fever, malaise and reduced appetite. On direct questioning, she did recall experiencing excessive overnight sweating on a few occasions but had not experienced joint pains. The patient denied other symptoms, including those associated with the respiratory, urinary tract and neurological systems.

The patient was otherwise healthy, with no significant past medical history. She took no regular medications and had no known medication allergies. The patient was a chartered accountant and lived with her husband and 5-year-old daughter. The patient had returned from a holiday in Turkey 4 weeks previously. During her holiday she had visited relatives in a rural village and recalled consuming local dairy products that she suspected had not been pasteurised.

On general examination, the patient appeared to be tired and pale. Examination of the patient's abdomen revealed palpable hepatomegaly and splenomegaly. Examinations of the respiratory, cardiovascular, neurological and musculoskeletal systems were unremarkable. Selected investigation results from the patient's emergency department attendance are given below.

Haemoglobin	117 g / dL
White cell count	3.9 x 10>3 / microlitre
Neutrophils	2.1 x 10>3 / microlitre (reference 1.8-7.8)
eGFR	> 90 ml / min / 1.73 m2
Alkaline phosphatase	134 U / L (reference 35-100)
ALT	57 U / L (reference 3-36)
Bilirubin	24 micromol / L (reference < 26)
C-reactive protein	68 mg / L (reference < 7)
Urine dipstick	No abnormality detected
Blood cultures	Small gram-positive coccobacilli; consistent with *Brucella* species
Chest x-ray	Clear lung fields; no evidence of consolidation or effusion

The likely diagnosis of Brucellosis was discussed with the patient. She expressed a very strong preference to receive treatment on an outpatient basis so that she would not be separated from her family.

What is the appropriate treatment regime?

- Doxycycline monotherapy for 2 weeks
- Proguanil with atovaquone for 3 days
- Clarithromycin and amoxicillin for 1 week
- Isoniazid, rifampicin, pyrazinamide and ethambutol for 8 weeks
- Doxycycline and rifampicin for 6 weeks

Submit answer

Reference ranges

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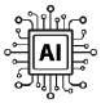
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Chest x-ray	Clear lung fields; no evidence of consolidation or effusion

The likely diagnosis of Brucellosis was discussed with the patient. She expressed a very strong preference to receive treatment on an outpatient basis so that she would not be separated from her family.

What is the appropriate treatment regime?

Doxycycline monotherapy for 2 weeks	43%
Proguanil with atovaquone for 3 days	1%
Clarithromycin and amoxicillin for 1 week	4%
Isoniazid, rifampicin, pyrazinamide and ethambutol for 8 weeks	1%
Doxycycline and rifampicin for 6 weeks	**51%**



Brucellosis is the most common bacterial disease contracted from contact with animals, most commonly sheep and goats. Infection can be acquired from consumption of unprocessed animal products in endemic countries of the Mediterranean and middle East such as Syria, Iraq, Turkey, Greece and Tunisia. Brucellosis can also be acquired by occupational exposure of laboratory workers or individuals with close contact with animals.

Brucellosis presents with non-specific symptoms and signs and so cases can go undiagnosed for several months. In addition to the typical symptoms and signs experienced by the patient in the question, brucellosis can also cause migratory arthritis, lymphadenopathy and malodorous perspiration. Rare complications include pneumonia, pulmonary effusion, peripheral neuropathy, meningoencephalitis and endocarditis. Positive blood cultures provide the most definitive diagnosis of brucellosis, although various serological tests are also available. Microbiological diagnosis is also possible from the culture of samples obtained from infected organs (for example, synovial fluid or cerebrospinal fluid). Brucellosis also causes non-specific abnormalities of routine blood tests as in the case of the patient in this question.

Oral treatment of brucellosis is possible with a 6-week course of doxycycline and rifampicin. An alternative treatment regime combines 6 weeks of oral doxycycline with 3 weeks of either intramuscular streptomycin or intravenous gentamicin. Treatment involving parenteral antibiotics has a lower failure rate but is less convenient for the patient.

The other possible answers are antimicrobial treatment regimes for *Helicobacter pylori*, tuberculosis, Lyme disease and malaria.

Ramin B, MacPherson P. Human brucellosis. *BMJ* 2010;341:c4545.

Brucellosis

Brucellosis is a zoonosis more common in the Middle East and in farmers, vets and abattoir workers. Four major species cause infection in humans: *B. melitensis* (sheep), *B. abortus* (cattle), *B. canis* and *B. suis* (pigs). Brucellosis has an incubation period 2 - 6 weeks.

Features

- non-specific: fever, malaise
- hepatosplenomegaly
- sacroiliitis: spinal tenderness may be seen
- complications: osteomyelitis, infective endocarditis, meningoencephalitis, orchitis
- leukopenia often seen

Diagnosis

- the Rose Bengal plate test can be used for screening but other tests are required to confirm the diagnosis
- Brucella serology is the best test for diagnosis
- blood and bone marrow cultures may be suitable in certain patients, but these tests are often negative

Management

- doxycycline and streptomycin

Textbooks

High-yield textbook

Extended textbook

Links

WHO

2 5

Brucellosis guidelines

Suggest link Report broken link

Score: **24%**

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A 35-year-old man is reviewed in respiratory clinic with symptoms of a chronic cough with occasional haemoptysis, night sweats and significant unintentional weight loss. The patient is originally from the Russian Federation and on direct questioning through an interpreter discloses that he had spent five years in a Moscow prison for drugs offences. Aside from a period of alcohol abuse in his early twenties, the patient denies any other medical history and takes no medications. He currently is living in shared accommodation and works on a building site, although he is struggling to continue to work due to his current illness.

General inspection showed the patient to be cachexic with tobacco stained fingernails. Auscultation of the chest revealed some reduced air entry in the upper zones bilaterally. There was palpable lymphadenopathy in the anterior cervical chain.

Please see results of investigations below.

Chest x-ray: patchy shadowing in both upper lobes with evidence of cavity formation on the right side; no pneumothorax; normal mediastinal shadow.

Sputum microscopy: acid fast bacilli

Phenotypic indirect drug susceptibility testing: rifampicin (resistant); ofloxacin (sensitive); moxifloxacin (sensitive); isoniazid (resistant); amikacin (sensitive); capreomycin (sensitive).

What is the most appropriate treatment regime for this patient?

- Combination therapy with 5 drugs for 24-36 months
- Combination therapy with 5 drugs for 18-24 months
- Combination therapy with 4 drugs for 24-36 months
- Combination therapy with 4 drugs for 12 months
- Combination therapy with 4 drugs for 18-24 months

Submit answer

Reference ranges

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What is the most appropriate treatment regime for this patient?

Combination therapy with 5 drugs for 24-36 months	5%
Combination therapy with 5 drugs for 18-24 months	41%
Combination therapy with 4 drugs for 24-36 months	4%
Combination therapy with 4 drugs for 12 months	32%
Combination therapy with 4 drugs for 18-24 months	18%

AI

The patient presents with a typical history for active pulmonary tuberculosis. His history of incarceration in a prison in the Russian Federation makes him at high risk of having contracted a multi-drug resistant strain of TB. Microbiology results confirm resistance to rifampicin and isoniazid consistent with multi-drug resistance, however without resistance to any fluoroquinolone

or second line injectable agents that would suggest extensively drug resistant TB.

Recommended treatment for multi-drug resistant TB requires 18-24 months of at least 5 drugs. Depending on the microbiological testing services available, the regime may be individualised to an individual patients infection or based on a standardised regime based on local resistance patterns.

Millard J, Ugarte-Gil C, Moore D. Multidrug resistant tuberculosis. BMJ 2015;350:h882.

Discuss (9) | Improve

Next question >

Tuberculosis: management

The standard therapy for treating **active tuberculosis** is:

Initial phase - first 2 months (RIPE)

- Rifampicin
- Isoniazid
- Pyrazinamide
- Ethambutol (the 2006 NICE guidelines now recommend giving a 'fourth drug' such as ethambutol routinely - previously this was only added if drug-resistant tuberculosis was suspected)

Continuation phase - next 4 months

- Rifampicin
- Isoniazid

The treatment for **latent tuberculosis** is 3 months of isoniazid (with pyridoxine) and rifampicin OR 6 months of isoniazid (with pyridoxine)

Patients with **meningeal tuberculosis** are treated for a prolonged period (at least 12 months) with the addition of steroids

Directly observed therapy with a three times a week dosing regimen may be indicated in certain groups, including:

- homeless people with active tuberculosis
- patients who are likely to have poor concordance
- all prisoners with active or latent tuberculosis

Complications of treatment

Immune reconstitution disease

- occurs typically 3-6 weeks after starting treatment
- often presents with enlarging lymph nodes

Drug adverse effects

- rifampicin
  - potent liver enzyme inducer
  - hepatitis, orange secretions
  - flu-like symptoms
- isoniazid
  - peripheral neuropathy: prevent with pyridoxine (Vitamin B6)
  - hepatitis, agranulocytosis
  - liver enzyme inhibitor
- pyrazinamide
  - hyperuricaemia causing gout
  - arthralgia, myalgia
  - hepatitis
- ethambutol
  - optic neuritis: check visual acuity before and during treatment



Next question

Textbooks

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Links

Cochrane

7 7

Corticosteroids for managing tuberculous meningitis

NICE 7 6

2016 Tuberculosis guidelines

Clinical Knowledge Summaries 8 3

Tuberculosis

Suggest link Report broken link

Media



Tuberculosis

Osmosis - YouTube 2 0

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Score: **24%**

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A 72-year-old man presents to the tuberculosis clinic.

He has a 6-month history of lower back pain that has been gradually getting worse. He has tried taking analgesia, but this is now no longer helping the pain and he is becoming less able to mobilise.

He lives with his wife on their farm. He has been fully independent until his recent deterioration in mobility.

Past medical history includes hypertension, diet-controlled type 2 diabetes mellitus and benign prostatic hypertrophy.

He reports that he had tuberculosis when he was 24, but this was treated. He can't remember what he was treated with.

An MRI spine shows a lumbar 4/5 discitis and a biopsy of this tissue is arranged. Acid-fast bacilli are grown on culture of the biopsy.

What treatment regime should be started?

- 2 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 4 months of isoniazid and rifampicin
- 6 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 6 months of isoniazid and rifampicin
- 6 months of isoniazid, rifampicin, pyrazinamide and ethambutol
- 2 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 7 months of isoniazid and rifampicin
- 3 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 6 months of isoniazid and rifampicin

Submit answer

Reference ranges

Score: **24%**

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2 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 4 months of isoniazid and rifampicin	37%
6 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 6 months of isoniazid and rifampicin	27%
6 months of isoniazid, rifampicin, pyrazinamide and ethambutol	4%
2 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 7 months of isoniazid and rifampicin	19%
3 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 6 months of isoniazid and rifampicin	13%

AI

Treatment for bone and joint tuberculosis is recommended to continue for 2 months with the initial phase consisting of quadruple therapy and the remaining 4 months of dual therapy.

It is recommended not to extend treatment for residual complications such as collapsed discs or bending of the spine, although there is some debate about this.

https://www.nice.org.uk/guidance/ng33/chapter/Recommendations#active-tb

Discuss (12) | Improve

Next question

Tuberculosis: management

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Next question

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Extended textbook

Links

Cochrane

7 7

Corticosteroids for managing tuberculous meningitis

NICE

7 6

2016 Tuberculosis guidelines

Clinical Knowledge Summaries

8 3

Tuberculosis

Suggest link Report broken link

Media



Tuberculosis

Osmosis - YouTube 2 0

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Back to top

A 45 year old man is diagnosed with HIV following attendance at Genitourinary clinic.

As part of his routine HIV work up his HLA B*5701 result is reported as positive.

Which drug will not be included in his ART regimen as a result of this information?

- Tenofovir
- Emtricitabine
- Lamivudine
- Stavudine
- Abacavir

Submit answer

Reference ranges

Score: **24%**

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Tenofovir	19%
Emtricitabine	21%
Lamivudine	16%
Stavudine	9%
Abacavir	**34%**

Abacavir can cause a severe and sometimes fatal hypersensitivity reaction. It is a cell mediated delayed hypersensitivity reaction which occurs around 6 weeks after starting treatment. Symptoms are of fever, rash, malaise and gastrointestinal symptoms.

Patients without HLA B*5701 are highly unlikely to develop hypersensitivity. Those with the HLA B*5701 allele have a 50% risk and therefore in these individuals abacavir should be avoided.

It is therefore routine to test HLA B*5701 status on all newly diagnosed patients with HIV.

Discuss (8) | Improve

Next question

HIV: management

Antiretroviral therapy (ART) involves a combination of at least three drugs, typically two nucleoside reverse transcriptase inhibitors (NRTI) and either a protease inhibitor (PI) or a non-nucleoside reverse transcriptase inhibitor (NNRTI). This combination both decreases viral replication but also reduces the risk of viral resistance emerging

Following the 2015 BHIVA guidelines it is now recommended that patients start ART as soon as they have been diagnosed with HIV, rather than waiting until a particular CD4 count, as was previously advocated.

Entry inhibitors

- maraviroc (binds to CCR5, preventing an interaction with gp41), enfuvirtide (binds to gp41, also known as a 'fusion inhibitor')
- prevent HIV-1 from entering and infecting immune cells

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), abacavir, emtricitabine, didanosine, lamivudine, stavudine, zalcitabine, tenofovir
- general NRTI side-effects: peripheral neuropathy
- tenofovir: used in BHIVAs two recommended regime NRTI. Adverse effects include renal impairment and ostesoporosis
- zidovudine: anaemia, myopathy, black nails
- didanosine: pancreatitis

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz
- side-effects: P450 enzyme interaction (nevirapine induces), rashes

Protease inhibitors (PI)

- examples: indinavir, nelfinavir, ritonavir, saquinavir
- side-effects: diabetes, hyperlipidaemia, buffalo hump, central obesity, P450 enzyme inhibition
- indinavir: renal stones, asymptomatic hyperbilirubinaemia
- ritonavir: a potent inhibitor of the P450 system

Integrase inhibitors

- block the action of integrase, a viral enzyme that inserts the viral genome into the DNA of the host cell
- examples: raltegravir, elvitegravir, dolutegravir

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

2016 HIV-1 infection guidelines

7 4

University of Liverpool

HIV Drug Interactions Checker

12 6

Suggest link

Report broken link

Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube

1 0



Antiretroviral drugs

Medinaz - YouTube

38 2

Report broken media

Score: **22%**

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Back to top

A 54 year old Caucasian man with HIV, is stable on Tenofovir, Emtricitabine and Kaltera (Lopinavir/Ritonavir). He has a 60 pack year smoking history and has been told his blood pressure is high previously but had never previously been to see his GP about it to get it treated. His GP is unaware of his HIV diagnosis and his main point of healthcare contact is with the Genito-urinary medicine services.

However over the last two years he has been troubled by gradually worsening cough, and shortness of breath. He has not lost weight. This has spurred him to start seeing his GP. He says that his GP initially gave him a blue inhaler and did some tests and told him he had COPD. Six weeks ago his GP gave him a Seretide 500 inhaler (one puff twice a day) and started him on Rampril (2.5mg daily) for his high blood pressure. The purple inhaler had provided some relief to his breathlessness but four days ago it ran out and he hasnt yet been back to see his GP. He still has two weeks worth of Ramipril tablets.

For the last few days he has been feeling faint and dizzy especially when standing up. His breathlessness and cough are no worse than usual. He has no fever. On examination his blood pressure is 91/76 his pulse is 94. He is afebrile, his respiratory rate is 24 and his oxygen saturations are 92% on room air. He has a mild wheeze on auscultation of his chest. His heart sounds are faint and his JVP is visible 3cm above his sternal angle. There is no pitting odema. There is nothing else of note on examination.

An ECG on admission is normal.

Hb	14.0 g/dl
Platelets	199 * 10^9/l
WBC	6.2 * 10^9/l

Na^+	130 mmol/l
K^+	5.9 mmol/l
Urea	5.6 mmol/l
Creatinine	85 µmol/l
CRP	7 mg/l

What is the diagnosis?

- Adrenal tuberculosis

- Pericardial tuberculosis
- Adrenal insufficiency secondary to inhaled corticosteroid withdrawal
- Ramipril induced hypotension
- WaterhouseFriderichsen syndrome

Submit answer

Reference ranges

Score: **24%**

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What is the diagnosis?

Adrenal tuberculosis	32%

Pericardial tuberculosis	11%
Adrenal insufficiency secondary to inhaled corticosteroid withdrawal	35%
Ramipril induced hypotension	7%
WaterhouseFriderichsen syndrome	15%

A very difficult question. The timing of events is key.

Fluticasone in Seretide is metabolised by P450 and Ritonvir inhibits this allowing very high levels of systemic steroid with consequent adrenal insufficiency. Cessation of Seretide has caused this Addisonian event (low sodium, high potassium is the clue).

Disseminated TB would be unusual with no fever or weight loss but is an important differential, especially in the context of HIV infection. All of his findings on examination are consistent with COPD.

Waterhouse-Friderichsen syndrome is bilateral adrenal haemorrhage in the context of meningococcal septicaemia.

Discuss (9) Improve

Next question

HIV: management

Antiretroviral therapy (ART) involves a combination of at least three drugs, typically two nucleoside reverse transcriptase inhibitors (NRTI) and either a protease inhibitor (PI) or a non-nucleoside reverse transcriptase inhibitor (NNRTI). This combination both decreases viral replication but also reduces the risk of viral resistance emerging

Following the 2015 BHIVA guidelines it is now recommended that patients start ART as soon as they have been diagnosed with HIV, rather than waiting until a particular CD4 count, as was previously advocated.

Entry inhibitors

- maraviroc (binds to CCR5, preventing an interaction with gp41), enfuvirtide (binds to gp41, also known as a 'fusion inhibitor')
- prevent HIV-1 from entering and infecting immune cells

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), abacavir, emtricitabine, didanosine, lamivudine, stavudine, zalcitabine, tenofovir
- general NRTI side-effects: peripheral neuropathy
- tenofovir: used in BHIVAs two recommended regime NRTI. Adverse effects include renal impairment and ostesoporosis
- zidovudine: anaemia, myopathy, black nails
- didanosine: pancreatitis

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz
- side-effects: P450 enzyme interaction (nevirapine induces), rashes

Protease inhibitors (PI)

- examples: indinavir, nelfinavir, ritonavir, saquinavir
- side-effects: diabetes, hyperlipidaemia, buffalo hump, central obesity, P450 enzyme inhibition
- indinavir: renal stones, asymptomatic hyperbilirubinaemia
- ritonavir: a potent inhibitor of the P450 system

Integrase inhibitors

- block the action of integrase, a viral enzyme that inserts the viral genome into the DNA of the host cell
- examples: raltegravir, elvitegravir, dolutegravir

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

7 4

2016 HIV-1 infection guidelines

University of Liverpool

12 6

HIV Drug Interactions Checker

Suggest link

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Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube

1 0



Antiretroviral drugs

Medinaz - YouTube

38 2

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Score: **22%**

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You see Mr Smith, a 35-year-old man-who-has-sex-with-men (MSM) in clinic. He was diagnosed with HIV 5 years ago, commencing combination antiretroviral therapy (cART). Following a number of alterations to his cART due to side effects he responded well to a combination of tenofovir, emtricitabine and ritonavir boosted atazanavir. His plasma viral load (pVL) of HIV RNA has remained undetectable and his adherence has been good.

In clinic today Mr Smith reports 5 weeks of drenching night sweats, a dry cough and some subjective weight loss, going up a belt buckle during this time period. On examination you note that he appears pale and auscultation of the chest elicits crepitations in the left upper zone. Your perform a chest X-ray which demonstrates a cavitating lesion in the left upper lobe. You arrange induced sputum samples which confirm a diagnosis of pulmonary tuberculosis.

You explain your diagnosis to Mr Smith and the need to urgently commence him on anti-tuberculosis chemotherapy. Whilst he is happy to commence treatment, he is adamant that he does not want to risk his viral control and states that he is not willing to consider altering his cART regime at present.

What is the most appropriate management step to treat Mr Smith?

- Hold his ART
- Commence rifabutin, isoniazid, ethambutol and pyrazinamide
- Commence isoniazid monotherapy
- Switch protease inhibitor to an novel ARV agent
- Commence rifampicin, isoniazid, pyrazinamide and ethambutol

Submit answer

Reference ranges

Score: **24%**

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What is the most appropriate management step to treat Mr Smith?

Hold his ART	3%
Commence rifabutin, isoniazid, ethambutol and pyrazinamide	47%
Commence isoniazid monotherapy	4%
Switch protease inhibitor to an novel ARV agent	13%
Commence rifampicin, isoniazid, pyrazinamide and ethambutol	32%

The correct answer for this scenario is to commence rifabutin, isoniazid, ethambutol and pyrazinamide.

This scenario tests your knowledge of rifamycins in the treatment of tuberculosis. Rifamycin containing anti-tuberculosis chemotherapy regimes are superior to those which do not contain a rifamycin agent. However, in this scenario the patient is also taking taking a protease inhibitor as part of their antiretroviral therapy.

Rifampicin, the common rifamycin agent in anti-tuberculosis regimes, is a potent inducer of liver

enzymes, such as cytochrome P450. Furthermore, it up-regulates the expression of P-glycoprotein in the gastrointestinal tract. This leads to reduced absorption and increased metabolism of certain medications, protease inhibitors being one of these (ritonavir booster atazanavir). Co-administration of a protease inhibitor with rifampicin therefore will often lead to subtherapeutic levels of the protease inhibitor.

In this scenario, the patient is stable on his current antiretroviral treatment and not keen to switch at present. Therefore, the British HIV Association (BHIVA) suggest the substitution of rifampicin for an alternative rifamycin agent (rifabutin or rifapentine), which has less inducing action of cytochrome P450 and therefore allows the patient to continue their current HIV treatment regime. Regimes containing rifabutin (dose 150mg three times a week) have been shown to have equivocal in outcomes of treatment in the few small observational studies performed in patients with HIV/TB co-infection.

Discuss (6) | Improve

Next question >

Tuberculosis: management

The standard therapy for treating **active tuberculosis** is:

Initial phase - first 2 months (RIPE)

- Rifampicin
- Isoniazid
- Pyrazinamide
- Ethambutol (the 2006 NICE guidelines now recommend giving a 'fourth drug' such as ethambutol routinely - previously this was only added if drug-resistant tuberculosis was suspected)

Continuation phase - next 4 months

- Rifampicin
- Isoniazid

The treatment for **latent tuberculosis** is 3 months of isoniazid (with pyridoxine) and rifampicin OR 6 months of isoniazid (with pyridoxine)

Patients with **meningeal tuberculosis** are treated for a prolonged period (at least 12 months) with the addition of steroids

Directly observed therapy with a three times a week dosing regimen may be indicated in certain groups, including:

- homeless people with active tuberculosis
- patients who are likely to have poor concordance
- all prisoners with active or latent tuberculosis

Complications of treatment

Immune reconstitution disease

- occurs typically 3-6 weeks after starting treatment
- often presents with enlarging lymph nodes

Drug adverse effects

- rifampicin
  - potent liver enzyme inducer
  - hepatitis, orange secretions
  - flu-like symptoms
- isoniazid
  - peripheral neuropathy: prevent with pyridoxine (Vitamin B6)
  - hepatitis, agranulocytosis
  - liver enzyme inhibitor
- pyrazinamide
  - hyperuricaemia causing gout
  - arthralgia, myalgia
  - hepatitis
- ethambutol
  - optic neuritis: check visual acuity before and during treatment



Next question

Textbooks

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Extended textbook

Links

Cochrane

Corticosteroids for managing tuberculous meningitis

7 7

NICE

2016 Tuberculosis guidelines

7 6

Clinical Knowledge Summaries

Tuberculosis

8 3

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Media



Tuberculosis

Osmosis - YouTube 2 0

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A 28-year-old man attends clinic for his 6 monthly HIV check. He was diagnosed with HIV 4 years ago and has been stable on Eviplera (emtricitabine/rilpivirine/tenofovir). He was recently started on a new medication by his GP.

Blood results are as follows:

Hb	134 g/l	Na^+	136 mmol/l
Platelets	345 * 10^9/l	K^+	4.2 mmol/l
CD4	580 * 10^9/l (normal range 500-1500)	Urea	6.5 mmol/l
Viral load	1480 copies/ml* 10^9/l	Creatinine	65 µmol/l

What new medication was started which would explain these results?

- Omeprazole
- Ciclosporin
- Tacrolimus
- Celecoxib
- Diclofenac

Submit answer

Reference ranges

Score: **24%**

1 ✓
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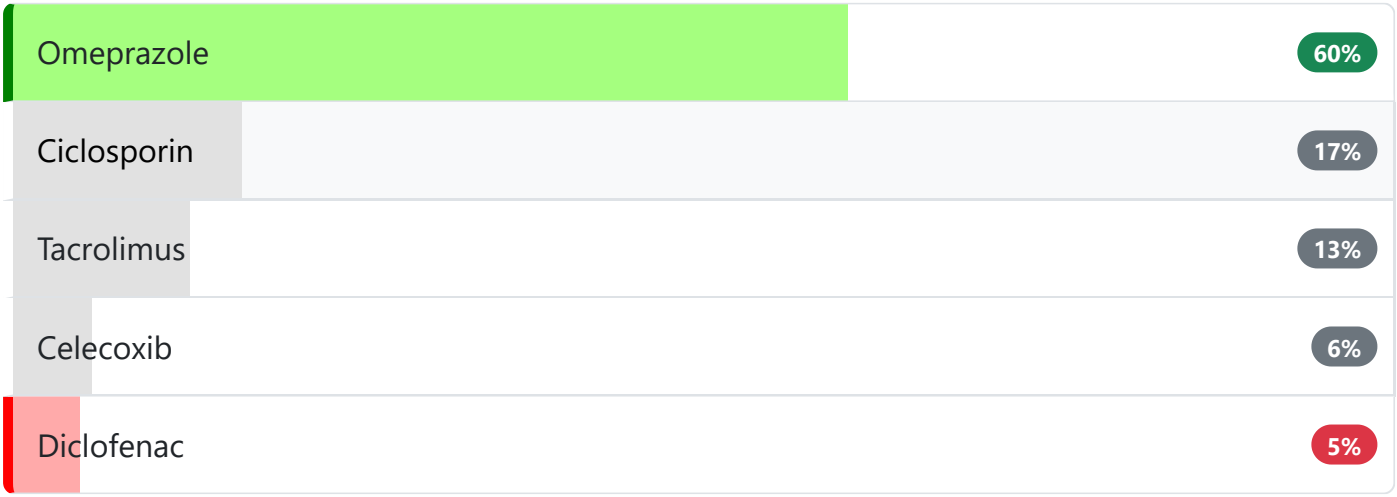
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PPIs are contraindicated in patients on Eviplera (emtricitabine/rilpivirine/tenofovir). They reduce the absorption of rilpivirine which can cause viral blips, with subsequent virological failure and resistance

Important for me Less important

Concurrent use of tenofovir with ciclosporin, tacrolimus, celecoxib or diclofenac, can increase the risk of nephrotoxicity.

Proton pump inhibitors are absolutely contraindicated in patients on Eviplera (emtricitabine/rilpivirine/tenofovir). They reduce the absorption of rilpivirine which can cause viral blips, with subsequent virological failure and resistance.

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123

Next question

Textbooks

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BHIVA

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2016 HIV-1 infection guidelines

University of Liverpool

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HIV Drug Interactions Checker

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Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube 1 0



Antiretroviral drugs

Medinaz - YouTube 38 2

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A 23-year-old medical student attends the emergency department after accidentally cutting herself in the kitchen, with significant blood loss. In addition to first aid for the wound, routine blood tests are checked. Results are as follows.

Hb	145 g/L	Male: (135-180) Female: (115 - 160)
Platelets	235 * 10^9/L	(150 - 400)
WBC	7.2 * 10^9/L	(4.0 - 11.0)
Eosinophils	0.91 * 10^9/L	(0.0-0.4)

Follow-up is organised in the infectious diseases clinic. The patient has recently returned from her elective in Malawi, where she spent 6 weeks with 2 friends. She reports taking all recommended vaccines before travel. Malaria prophylaxis was taken as advised and she completed the course. She denies swimming in any water but did frequently walk barefoot around the banks of the lake. She recalls one episode where her foot became itchy and developed a serpiginous rash, advancing up the foot over several hours before resolving. She did not seek medical help.

What treatment is recommended first-line for this condition?

- Artemisinin combination therapy
- Ivermectin
- Melarsoprol
- Nifurtimox/eflornithine combination therapy
- Praziquantel

Submit answer

Reference ranges

Score: **24%**

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What treatment is recommended first-line for this condition?

Artemisinin combination therapy	1%
Ivermectin	71%
Melarsoprol	2%
Nifurtimox/eflornithine combination therapy	2%
Praziquantel	24%

Ivermectin is the drug of choice for treatment of strongyloidiasis

Important for me Less important

The correct answer is **ivermectin**. This patient is likely to have developed strongyloidiasis, a parasitic infection caused by the nematode Strongyloides stercoralis. This is a widely-distributed helminth but the majority of cases are seen in sub-Saharan Africa and South America. Infection is frequently asymptomatic and the first clue is peripheral eosinophilia. In this case, the episode

described in Malawi is consistent with larva currens, a rash that develops when the worm penetrates the skin before entering the bloodstream. From here they spread through the lungs, over the epiglottis, are swallowed, and settle in the small intestine, where they lay their eggs. Respiratory symptoms are rare but cough and wheeze may develop during pulmonary migration. In chronic infection, abdominal pain and diarrhoea may occur. In the context of immunocompromise, Strongyloides hyperinfection syndrome can occur, presenting with bloody colitis and pneumonitis, often with pleural effusions. The treatment of choice in all cases is ivermectin, although albendazole and thiabendazole are alternatives.

Artemisinin combination therapy is incorrect. This is the treatment of choice for most cases of uncomplicated malaria. Malaria does not tend to cause eosinophilia or skin lesions, and she has been compliant with prophylaxis. These factors suggest this is very unlikely to be the diagnosis.

Melarsoprol is incorrect. This is an arsenic-based treatment option for East African sleeping sickness, caused by Trypanosoma brucei rhodesiense. This is a rapidly progressive form of sleeping sickness, initially causing an influenza-like illness with arthralgia and lymphadenopathy but quickly progressing to altered mental status and death. Melarsoprol is associated with significant toxicity and would not be used for strongyloidiasis.

Nifurtimox/eflornithine combination therapy is incorrect. This is a treatment option for West African sleeping sickness, caused by the protozoan parasite Trypanosoma brucei gambiense. This causes a slowly progressive illness characterised by behavioural change, alterations to the sleep-wake cycle, and finally coma. There are no symptoms here to suggest this and it is not typically associated with eosinophilia.

Praziquantel is incorrect. This is the treatment of choice for schistosomiasis, a tapeworm infection caused by a variety of species of schistosomes. The organism uses freshwater snails as an intermediate host before being released into the water, where it penetrates human skin and settles in the gastrointestinal or genito-urinary tract depending on the species. There may be an itchy rash at the time of infection, known as cercarial dermatitis, but this would not move up the limb or be described as serpiginous. Schistosomiasis often presents with eosinophilia, but in this case, the patient has been careful to avoid going in the water, making this unlikely to be the cause.

Discuss (7) | Improve

Next question >

Strongyloides stercoralis

Strongyloides stercoralis is a human parasitic nematode worm. The larvae are present in soil and gain access to the body by penetrating the skin. Infection with *Strongyloides stercoralis* causes strongyloidiasis.

Features

- diarrhoea
- abdominal pain/bloating
- papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks
- larva currens: pruritic, linear, urticarial rash
- if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered

Treatment

- ivermectin and albendazole are used

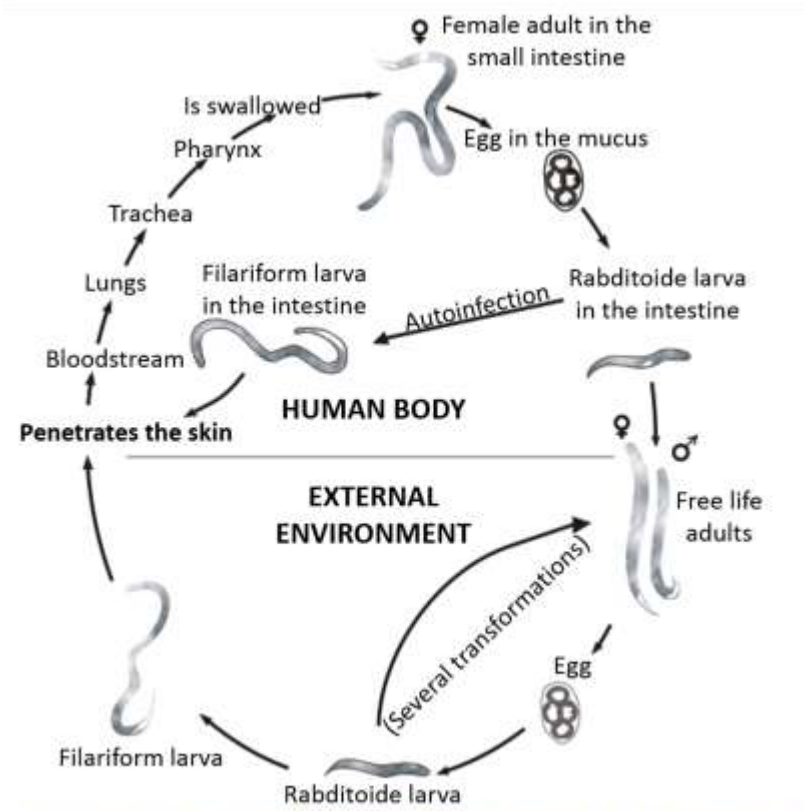


Image sourced from Wikipedia

Diagram showing the lifecycle of *Strongyloides stercoralis*

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **22%**

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A 53-year-old lady has been admitted under isolation to a tertiary infectious diseases centre in London. She has recently arrived back to the United Kingdom following 2 years of ecological work in lowland Nepal and has presented with 4 weeks of worsening malaise, fevers, night sweats, pallor and abdominal pain. She had lived during her time in Nepal in a small village where she reported numerous fly bites during the evening hours. She reports taking her malaria prophylaxis consistently.

Examination shows her to look pale, unwell and her skin appears to have an unnatural grey tinge. No rash nor joint swelling was noted. She has a temperature of 37.9ºC, oxygen saturation 97% on air, heart rate 84 beats per minute, respiratory rate 23 breaths per minute and blood pressure 128/75 mmHg. Auscultation of the chest reveals bilateral vesicular breath sounds and her heart sounds are normal with no murmurs. Abdominal examination reveals a soft abdomen with tenderness in the upper quadrants. Her liver edge is palpable 5cm below the right costal margin and an additional mass is palpable in the umbilical area which moves with ventilation.

Investigation results are as follows:

Hb	102 g/L	Female: (115 - 160)
Platelets	134 * 10^9/L	(150 - 400)
WBC	3.6 * 10^9/L	(4.0 - 11.0)
Na^+	144 mmol/L	(135 - 145)
K^+	3.7 mmol/L	(3.5 - 5.0)
Urea	6.3 mmol/L	(2.0 - 7.0)
Creatinine	105 µmol/L	(55 - 120)
CRP	73 mg/L	(< 5)

Thick blood film	No evidence of malaria parasites
HIV 1 and 2 surface antigen/antibodies	Negative
Hepatitis A, B, C, D and E	Negative
Chest X-ray	No focal consolidation, masses or collapse
quantiFERON testing	Negative

Which of the following investigation would be the gold standard to confirm the most likely diagnosis?

- Direct agglutination test (DAT)

- Lumbar puncture and CSF analysis
- Stool sample analysis
- Blood cultures
- Splenic aspirate

Submit answer

Reference ranges

Score: **24%**

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Thick blood film	No evidence of malaria parasites
HIV 1 and 2 surface antigen/antibodies	Negative
Hepatitis A, B, C, D and E	Negative
Chest X-ray	No focal consolidation, masses or collapse
quantiFERON testing	Negative

Which of the following investigation would be the gold standard to confirm the most likely diagnosis?

Direct agglutination test (DAT) 13%

Lumbar puncture and CSF analysis	4%
Stool sample analysis	14%
Blood cultures	12%
Splenic aspirate	57%

Demonstration of parasites on bone marrow or splenic aspirate remains gold standard for diagnosing visceral leishmaniasis

Important for me Less important

Leishmaniasis is spread through sand-fly vectors from animal reservoirs and remains endemic through areas of Africa, Asia, South America and the Middle East. Visceral leishmaniasis can cause hepatosplenomegaly, spiking fevers, pancytopenia and with certain strains blackening of the skin leading to the alternative name of black fever (kala-azar). The gold standard for diagnosis would be the demonstration of amastigotes on splenic or bone marrow aspirates.

Serological testing, such as DAT, is useful in the diagnosis of leishmaniasis but it is not the gold standard due to lack of evidence demonstrating consistently higher sensitivities and specificities along with a regional variation of the parasites complicating this. They are however highly useful in endemic areas where it is a cheaper, less technically challenging and occasionally a readily available alternative.

Lumbar puncture and CSF analysis are not useful investigations in the diagnosis of leishmaniasis unless an alternative or additional pathology is suspected.

A stool sample is of no use in the diagnosis of leishmaniasis which is a vector born disease transmitted through sandfly bites.

Blood cultures can be useful in demonstrating blood-born parasites in immunocompetent hosts with sensitivity rates up to 60%. This, however, remains below that of the current gold standard.

Discuss (9) Improve

Next question ›

Leishmaniasis

Leishmaniasis is caused by the intracellular protozoa *Leishmania*, which are spread by the bites of sandflies. Cutaneous, mucocutaneous leishmaniasis and visceral forms are seen

Cutaneous leishmaniasis

- caused by *Leishmania tropica* or *Leishmania mexicana*
- crusted lesion at the site of bite
- there may be an underlying ulcer
- it is typically diagnosed by doing a punch biopsy from the edge of the lesion allowing for both histology and culture
- cutaneous leishmaniasis acquired in South or Central America merits treatment due to the risk of mucocutaneous leishmaniasis whereas disease acquired in Africa or India can be managed more conservatively







Mucocutaneous leishmaniasis

- caused by *Leishmania braziliensis*
- skin lesions may spread to involve mucosae of nose, pharynx etc

Visceral leishmaniasis (kala-azar)

- mostly caused by *Leishmania donovani*
- occurs in the Mediterranean, Asia, South America, Africa
- fever, sweats, rigors
- massive splenomegaly. hepatomegaly
- poor appetite*, weight loss
  - occasionally patients may report increased appetite with paradoxical weight loss
- grey skin - 'kala-azar' means black sickness
- pancytopaenia secondary to hypersplenism
- the gold standard for diagnosis is bone marrow or splenic aspirate
- management
  - the BNF recommends sodium stibogluconate, an organic pentavalent antimony compound
  - amphotericin B may be used with or after treatment with an antimony compound

AI 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 9 8

2011 Leishmaniasis review

DermNet NZ 9 6

Leishmaniasis review

Suggest link Report broken link

Media



What is Leishmaniasis? An introduction and overview

Let's Learn About Bugs - YouTube 5 2

Report broken media

Score: **22%**

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A 45-year-old homeless gentleman presents with a two-month history of a cough, occasional haemoptysis and weight loss. His chest X-ray shows right upper lobe consolidation. A diagnosis of tuberculosis is confirmed by microscopy and he is started empirically on rifampicin, isoniazid, pyrazinamide and ethambutol, with pyridoxine supplements. Eight weeks later, culture results show that the Mycobacterium tuberculosis is resistant to isoniazid. What should his further treatment regime be?

- Four months of rifampicin, isoniazid, pyrazinamide and ethambutol, with pyridoxine supplements
- Six months of rifampicin
- Four months of rifampicin and ethambutol, with pyridoxine supplements
- Four months of rifampicin and ethambutol
- Add a quinolone and aminoglycoside

Submit answer

Reference ranges

Score: **24%**

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Four months of rifampicin, isoniazid, pyrazinamide and ethambutol, with pyridoxine supplements	7%
Six months of rifampicin	7%
Four months of rifampicin and ethambutol, with pyridoxine supplements	16%
Four months of rifampicin and ethambutol	24%
Add a quinolone and aminoglycoside	46%

In confirmed isoniazid resistance, ethambutol is continued for 6 months of treatment

Important for me Less important

In fully sensitive Mycobacterium tuberculosis infection, after two months, the pyrazinamide and ethambutol are stopped and rifampicin and isoniazid (with pyridoxine support) are continued for a further four months. However, in cases of confirmed isoniazid resistance, rifampicin and ethambutol are continued for the further 4 months of treatment, with discontinuation of the isoniazid (and supportive pyridoxine) and pyrazinamide.

This is not multi-drug resistant tuberculosis (does not respond to at least isoniazid and rifampicin), so it is not appropriate to add a quinolone or aminoglycoside.

Discuss (8) Improve

Next question

Tuberculosis: management

The standard therapy for treating **active tuberculosis** is:

Initial phase - first 2 months (RIPE)
- Rifampicin
- Isoniazid
- Pyrazinamide
- Ethambutol (the 2006 NICE guidelines now recommend giving a 'fourth drug' such as ethambutol routinely - previously this was only added if drug-resistant tuberculosis was suspected)

Continuation phase - next 4 months
- Rifampicin
- Isoniazid

The treatment for **latent tuberculosis** is 3 months of isoniazid (with pyridoxine) and rifampicin OR 6 months of isoniazid (with pyridoxine)

Patients with **meningeal tuberculosis** are treated for a prolonged period (at least 12 months) with the addition of steroids

Directly observed therapy with a three times a week dosing regimen may be indicated in certain groups, including:
- homeless people with active tuberculosis
- patients who are likely to have poor concordance
- all prisoners with active or latent tuberculosis

Complications of treatment

Immune reconstitution disease
- occurs typically 3-6 weeks after starting treatment
- often presents with enlarging lymph nodes

Drug adverse effects
- rifampicin
  - potent liver enzyme inducer
  - hepatitis, orange secretions
  - flu-like symptoms
- isoniazid
  - peripheral neuropathy: prevent with pyridoxine (Vitamin B6)
  - hepatitis, agranulocytosis
  - liver enzyme inhibitor

- pyrazinamide
  - hyperuricaemia causing gout
  - arthralgia, myalgia
  - hepatitis
- ethambutol
  - optic neuritis: check visual acuity before and during treatment

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Cochrane 7 7

Corticosteroids for managing tuberculous meningitis

NICE 7 6

2016 Tuberculosis guidelines

Clinical Knowledge Summaries 8 3

Tuberculosis

Suggest link Report broken link

Media



Tuberculosis

Osmosis - YouTube 2 0

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Score: **22%**

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A 22 year old male attends the Genitourinary Medicine clinic which you are working in. He was contacted and asked to attend having been in direct, recent sexual contact with gonorrhoea. He is asymptomatic. What is the best course of action?

- No action required as he is asymptomatic
- Treat with ceftriaxone, perform a sexual health screen and call back for a test of cure if positive
- Give ceftriaxone and azithromycin, no need for a screen
- Perform a screen and call back to treat if positive
- Give ceftriaxone, perform a screen, and call back to give azithromycin if positive

Submit answer

Reference ranges

Score: **24%**

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A 22 year old male attends the Genitourinary Medicine clinic which you are working in. He was contacted and asked to attend having been in direct, recent sexual contact with gonorrhoea. He is asymptomatic. What is the best course of action?

No action required as he is asymptomatic	3%
Treat with ceftriaxone, perform a sexual health screen and call back for a test of cure if positive	59%
Give ceftriaxone and azithromycin, no need for a screen	7%
Perform a screen and call back to treat if positive	17%
Give ceftriaxone, perform a screen, and call back to give azithromycin if positive	14%

The British Association of Sexual Health and HIV have clear guidelines on the investigation and management of gonorrhoea.

Recent sexual contact with person in whom gonococcal infection has been confirmed is an indication for treatment.

Treatment of uncomplicated gonorrhoea infection consists of ceftriaxone intramuscularly as a single dose. Due to increased resistance to a range of antimicrobials in recent years follow up 'test of cure' is recommended in all confirmed cases.

Discuss (7) | Improve

Next question

Gonorrhoea

Gonorrhoea is caused by the Gram-negative diplococcus *Neisseria gonorrhoeae*. Acute infection can occur on any mucous membrane surface, typically genitourinary but also rectum and pharynx. The incubation period of gonorrhoea is 2-5 days

Features

- males: urethral discharge, dysuria
- females: cervicitis e.g. leading to vaginal discharge
- rectal and pharyngeal infection is usually asymptomatic

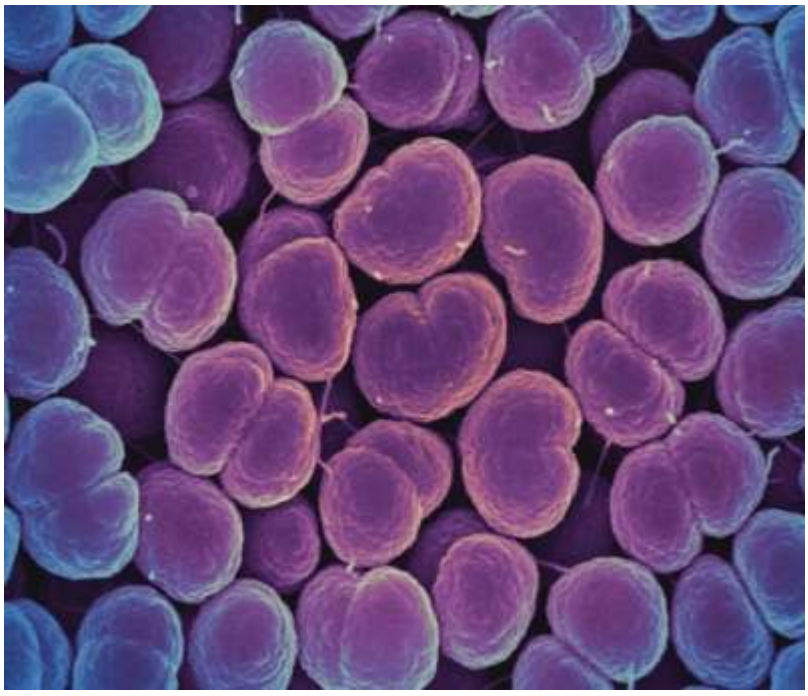
Microbiology

- immunisation is not possible and reinfection is common due to antigen variation of type IV pili (proteins which adhere to surfaces) and Opa proteins (surface proteins which bind to receptors on immune cells)

Local complications that may develop include urethral strictures, epididymitis and salpingitis (hence may lead to infertility). Disseminated infection may occur - see below

Management

- ciprofloxacin used to be the treatment of choice. However, there is increased resistance to ciprofloxacin (around 36% in the UK) and therefore cephalosporins are now more widely used
- there was a change in the 2019 British Society for Sexual Health and HIV (BASHH) guidelines. Previously the first-line treatment was IM ceftriaxone + oral azithromycin. The new first-line treatment is a single dose of IM ceftriaxone 1g (i.e. no longer add azithromycin). If sensitivities are known (and the organism is sensitive to ciprofloxacin) then a single dose of oral ciprofloxacin 500mg should be given
- if ceftriaxone is refused (e.g. needle-phobic) then oral cefixime 400mg (single dose) + oral azithromycin 2g (single dose) should be used



Colorized scanning electron micrograph of *Neisseria gonorrhoeae*. Credit: NIAID

Disseminated gonococcal infection (DGI) and gonococcal arthritis may also occur, with gonococcal infection being the most common cause of septic arthritis in young adults. The pathophysiology of

DGI is not fully understood but is thought to be due to haematogenous spread from mucosal infection (e.g. Asymptomatic genital infection). Initially there may be a classic triad of symptoms: tenosynovitis, migratory polyarthritis and dermatitis. Later complications include septic arthritis, endocarditis and perihepatitis (Fitz-Hugh-Curtis syndrome)

Key features of disseminated gonococcal infection

- tenosynovitis
- migratory polyarthritis
- dermatitis (lesions can be maculopapular or vesicular)



Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Society for Sexual Health and HIV (BASHH) 11 6

2019 Gonorrhoea guidelines

Clinical Knowledge Summaries 10 3

Gonorrhoea guidelines

Suggest link Report broken link

Media



Neisseria gonorrhoeae

Osmosis - YouTube

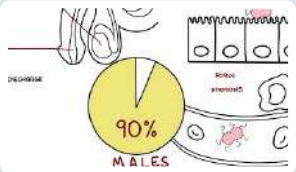
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Neisseria gonorrhoeae

ATP - YouTube

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Neisseria gonorrhea (STI) symptoms, pathophysiology, complications and treatment

Armando Hasudungan - YouTube

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Score: **22%**

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A 31 year-old accountant was referred to the general medical clinic with persistent nasal stuffiness and intermittent epistaxis. He had been seen by the ear, nose and throat team who had commenced him on a course of intranasal steroids without benefit. His past medical history was unremarkable. His travel history included a gap year in South America 10 years previously. He was a non-smoker and drank 23 units of alcohol per week.

On examination, his temperature was 36.2ºC, heart rate 68 beats per minute, respiratory rate 16 breaths per minute, blood pressure 126/82 mmHg. There was some superficial ulceration of the nasal mucosa bilaterally. The chest was clear on auscultation and heart sounds were normal.

Investigations:

Haemoglobin	145 g/L
White cell count	7.0 * 10^9/l
Neutrophil count	4.0 * 10^9/l
Lymphocyte count	3.0 * 10^9/l
Eosinophil count	0.4 * 10^9/l
Platelets	300 * 10^9/l

Sodium	144 mmol/L
Potassium	4.1 mmol/L
Urea	6.1 mmol/L
Creatinine	71 mol/L
Alkaline phosphatase	56 IU/L
Alanine aminotransferase	35 IU/L
Gamma-glutyl transferase	21 IU/L
Bilirubin	13 mol/L
Albumin	39 g/L
Fasting plasma glucose	5.3 mmol/L

What is the most likely causative organism?

- *Trypanosoma cruzi*
- *Plasmodium ovale*

- ○ *Leishmania braziliensis* ×
- ○ *Leishmania donovani* ×
- ○ *Chlamydia trachomatis* ×

Submit answer

Reference ranges

Score: **24%**

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Gamma-glutyl transferase	21 IU/L
Bilirubin	13 mol/L
Albumin	39 g/L
Fasting plasma glucose	5.3 mmol/L

What is the most likely causative organism?

Trypanosoma cruzi	12%
Plasmodium ovale	1%

Leishmania braziliensis	69%
Leishmania donovani	14%
Chlamydia trachomatis	3%

The most likely diagnosis in this case is mucosal leishmaniasis. The patient probably became infected with cutaneous leishmaniasis during his visit to South America, and received no/incomplete treatment. Mucosal leishmaniasis can occur years to decades after initial infection; typical features include nasal stuffiness and epistaxis in the early stages, progressing to ulceration and destruction of the nasal mucosa. *Leishmania donovani* is a recognised cause of visceral rather than mucocutaneous leishmaniasis.

Discuss (5) | Improve

Next question

Leishmaniasis

Leishmaniasis is caused by the intracellular protozoa *Leishmania*, which are spread by the bites of sandflies. Cutaneous, mucocutaneous leishmaniasis and visceral forms are seen

Cutaneous leishmaniasis

- caused by *Leishmania tropica* or *Leishmania mexicana*
- crusted lesion at the site of bite
- there may be an underlying ulcer
- it is typically diagnosed by doing a punch biopsy from the edge of the lesion allowing for both histology and culture
- cutaneous leishmaniasis acquired in South or Central America merits treatment due to the risk of mucocutaneous leishmaniasis whereas disease acquired in Africa or India can be managed more conservatively





Mucocutaneous leishmaniasis

- caused by *Leishmania braziliensis*
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Visceral leishmaniasis (kala-azar)

- mostly caused by *Leishmania donovani*
- occurs in the Mediterranean, Asia, South America, Africa
- fever, sweats, rigors
- massive splenomegaly. hepatomegaly
- poor appetite*, weight loss
    - occasionally patients may report increased appetite with paradoxical weight loss
- grey skin - 'kala-azar' means black sickness
- pancytopaenia secondary to hypersplenism
- the gold standard for diagnosis is bone marrow or splenic aspirate
- management
    - the BNF recommends sodium stibogluconate, an organic pentavalent antimony compound
    - amphotericin B may be used with or after treatment with an antimony compound



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians

9 8

2011 Leishmaniasis review

DermNet NZ 9 6

Leishmaniasis review

Suggest link Report broken link

Media



What is Leishmaniasis? An introduction and overview

Let's Learn About Bugs - YouTube 5 2

Report broken media

Score: **22%**

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Back to top

A 30-year-old male with HIV and a viral load of 10^6 and CD4 dropping from 500 to 300 cells/µl since his last visit. The above mentioned results are taken while he is on his third line of therapy. What is the most likely cause of his repeated treatment failures?

- Non-compliance
- Infection with multi-resistant HIV strain
- Co-infection with a new HIV strain
- Superinfection with a new HIV strain
- CYP interactions

Submit answer

Reference ranges

Score: **24%**

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Back to top

A 30-year-old male with HIV and a viral load of 10^6 and CD4 dropping from 500 to 300 cells/µl since his last visit. The above mentioned results are taken while he is on his third line of therapy. What is the most likely cause of his repeated treatment failures?

Non-compliance	58%
Infection with multi-resistant HIV strain	18%
Co-infection with a new HIV strain	4%
Superinfection with a new HIV strain	5%
CYP interactions	14%

AI

By far the most common cause of treatment failure in HIV therapy is treatment non-compliance.

Re-infection with new HIV strain can occur via co-infection or superinfection. Co-infection is defined as infection with 2 strains either simultaneously or within a brief period of time before infection with the first strain has an established immune response. Arbitrarily, HIV coinfection would occur within the first month or so of infection. Superinfection is defined as infection with a second strain after the initial infection and the immune response to it has been established.

Discuss (5) | Improve

Next question >

HIV: management

Antiretroviral therapy (ART) involves a combination of at least three drugs, typically two nucleoside reverse transcriptase inhibitors (NRTI) and either a protease inhibitor (PI) or a non-nucleoside reverse transcriptase inhibitor (NNRTI). This combination both decreases viral replication but also reduces the risk of viral resistance emerging

Following the 2015 BHIVA guidelines it is now recommended that patients start ART as soon as they have been diagnosed with HIV, rather than waiting until a particular CD4 count, as was previously advocated.

Entry inhibitors

- maraviroc (binds to CCR5, preventing an interaction with gp41), enfuvirtide (binds to gp41, also known as a 'fusion inhibitor')
- prevent HIV-1 from entering and infecting immune cells

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), abacavir, emtricitabine, didanosine, lamivudine, stavudine, zalcitabine, tenofovir
- general NRTI side-effects: peripheral neuropathy
- tenofovir: used in BHIVAs two recommended regime NRTI. Adverse effects include renal impairment and ostesoporosis
- zidovudine: anaemia, myopathy, black nails
- didanosine: pancreatitis

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz
- side-effects: P450 enzyme interaction (nevirapine induces), rashes

Protease inhibitors (PI)

- examples: indinavir, nelfinavir, ritonavir, saquinavir
- side-effects: diabetes, hyperlipidaemia, buffalo hump, central obesity, P450 enzyme inhibition
- indinavir: renal stones, asymptomatic hyperbilirubinaemia
- ritonavir: a potent inhibitor of the P450 system

Integrase inhibitors

- block the action of integrase, a viral enzyme that inserts the viral genome into the DNA of the host cell
- examples: raltegravir, elvitegravir, dolutegravir



Next question

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

7 4

2016 HIV-1 infection guidelines

University of Liverpool

12 6

HIV Drug Interactions Checker

Suggest link

Report broken link

Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube

1 0



Antiretroviral drugs

Medinaz - YouTube

38 2

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Score: **22%**

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A 23-year-old man presents to the emergency department with a fever and a rash having recently returned back from a back-packing trip in rural Thailand where he had been trekking. He complains of general malaise for the last few days, a frontal headache and a rash he has noticed from last night. On examination he had tender cervical lymphadenopathy and a maculopapular rash over his trunk. There was also a small painless erythematous lesion on his lower calf with a necrotic centre.

Hb	95 g/l
Platelets	60 * 10^9/l
WBC	2.5 * 10^9/l

What is the most likely diagnosis?

- Malaria
- Dengue fever
- Scrub typhus
- Leptospirosis
- HIV conversion illness

Submit answer

Reference ranges

Score: **24%**

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Hb	95 g/l
Platelets	60 * 10^9/l
WBC	2.5 * 10^9/l

What is the most likely diagnosis?

Malaria	1%
Dengue fever	19%
Scrub typhus	72%
Leptospirosis	4%
HIV conversion illness	4%

The eschar makes scrub typhus the most likely diagnosis. Malaria is of course common in this region and one would expect similar laboratory results, however the rash makes this diagnosis very unlikely. Dengue fever presents with a rash and fever, but also with associated arthropathy, classically lower back pain.

Discuss (4) | Improve

Next question >

Typhus

Typhus is a group of rickettsial diseases transmitted between hosts by arthropods. Examples include

- endemic typhus
  - caused by *Rickettsia typhi*
  - reservoir/vector: fleas on rats
  - occurs throughout the world, typically in warm costal regions
  - relative bradycardia
- epidemic typhus
  - caused by *Rickettsia prowazekii*
  - reservoir/vector: body louse
  - more common in central and eastern Africa, central and South America
- scrub typhus
  - caused by *Orientia tsutsugamushi*
  - reservoir/vector: harvest mites on humans or rodents
  - more common in Asia
  - black eschar at site of original inoculation
  - relative bradycardia despite fever
- spotted fever
  - caused by Rickettsia spotted fever group
  - spread by ticks
  - examples include Rocky Mountain spotted fever

Common features

- fever, headache, malaise
- rash
  - typically maculopapular
  - begins on the trunk and spreads to the extremities
- later complications
  - meningoencephalitis

Management

- doxycycline



Next question

Textbooks

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Score: **22%**

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A 40-year-old-year presents to the outpatient department with three separate lesions on his hands. He reports the skin lesions developed over several months and does not remember an initial trigger or injury resulting in the nodular-like ulcers.

The patient reports he has just arrived from rural Tanzania where he has lived most of his life. He is HIV positive on treatment but has not attended any recent clinic appointments. He reports he has no other medical conditions and is not on any other medications.

Fine needle aspiration of the lesions shows the presence of protozoa particles.

A picture of one of the patient's lesions is shown below.



© Image used on license from DermNet NZ

What is the most likely cause of the patient's skin lesion?

- African trypanosomiasis
- Chromoblastomycosis
- Kaposi's sarcoma
- Leishmaniasis
- Leprosy

Submit answer

Reference ranges

Score: **24%**

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Fine needle aspiration of the lesions shows the presence of protozoa particles.

A picture of one of the patient's lesions is shown below.



© Image used on license from DermNet NZ

What is the most likely cause of the patient's skin lesion?

African trypanosomiasis	12%
Chromoblastomycosis	3%
Kaposi's sarcoma	4%
Leishmaniasis	79%
Leprosy	2%

Leishmaniasis is caused by the protozoa parasite *Leishmania tropica* or *Leishmania mexicana* which are transmitted via sandfly mainly in the tropics and sub-tropics of Africa, Asia and the Americas. Cutaneous leishmaniasis is the commonest form of leishmaniasis, which presents an open sore at the bite sites. The lesions can develop over several months or even years leaving unpleasant-looking scars.

African trypanosomiasis is caused by the protozoa parasite *Trypanosoma bruce* which is transmitted via tsetse flies. The condition causes systemic symptoms in the form initially of fevers, headaches and joint pain, before progressing to cognitive impairment. Cutaneous lesions are not commonly associated with the condition.

Chromoblastomycosis is normally found in rural region of tropical and subtropical climates however it is a chronic fungal infection of the skin. It can be caused by a number of different fungi that are implanted under the skin, often by thorns or splinters. Analysis of skin scraping would confirm the presence of muriform cells, indicative of the fungal infection.

Kaposi's sarcoma (KS) is a type of cancer that can form nodular, blotchy-like lesions red, purple, brown, or black in colour on the skin of the lower limbs, back, face and genitals, as well as mouth and organs lesions. KS is a result of immune suppression and infection by human herpesvirus 8; it is an aids defining condition and therefore is normally seen in untreated HIV. Diagnosis is via confirming the presents of human herpesvirus 8.

Leprosy is caused by the slow-growing, acid-fast bacillus, *Mycobacterium leprae* and is transmitted by humans via respiratory droplets. Diagnosis is confirmed via a skin smear from a lesion that is stained for mycobacteria.

Discuss (2) | Improve

Next question >

Leishmaniasis ★

Leishmaniasis is caused by the intracellular protozoa *Leishmania*, which are spread by the bites of sandflies. Cutaneous, mucocutaneous leishmaniasis and visceral forms are seen

Cutaneous leishmaniasis

- caused by *Leishmania tropica* or *Leishmania mexicana*
- crusted lesion at the site of bite
- there may be an underlying ulcer
- it is typically diagnosed by doing a punch biopsy from the edge of the lesion allowing for both histology and culture

- cutaneous leishmaniasis acquired in South or Central America merits treatment due to the risk of mucocutaneous leishmaniasis whereas disease acquired in Africa or India can be managed more conservatively





Mucocutaneous leishmaniasis

- caused by *Leishmania braziliensis*
- skin lesions may spread to involve mucosae of nose, pharynx etc

Visceral leishmaniasis (kala-azar)

- mostly caused by *Leishmania donovani*
- occurs in the Mediterranean, Asia, South America, Africa
- fever, sweats, rigors
- massive splenomegaly. hepatomegaly
- poor appetite*, weight loss
    - occasionally patients may report increased appetite with paradoxical weight loss
- grey skin - 'kala-azar' means black sickness
- pancytopaenia secondary to hypersplenism
- the gold standard for diagnosis is bone marrow or splenic aspirate
- management
    - the BNF recommends sodium stibogluconate, an organic pentavalent antimony compound
    - amphotericin B may be used with or after treatment with an antimony compound

AI + Q 123

Next question >

B I A T↕

Textbooks

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Links

Royal College of Physicians 9 8

2011 Leishmaniasis review

DermNet NZ 9 6

Leishmaniasis review

Suggest link Report broken link

Media



What is Leishmaniasis? An introduction and overview

Let's Learn About Bugs - YouTube 5 2

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An elderly gentleman presents with a three day history of bloody diarrhoea and feverishness. He has no significant travel history. His past medical history is listed as hypertension, osteoarthritis and gout. On examination his temperature is 38.0ºC, heart rate 95/min, blood pressure 120/80 mmHg and his abdomen is soft and non-tender. A stool sample has grown *Salmonella*. What is the best treatment?

- Metronidazole
- Doxycycline
- Clarithromycin
- Ciprofloxacin
- Amoxicillin

Submit answer

Reference ranges

Score: **24%**

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Metronidazole	7%
Doxycycline	5%
Clarithromycin	11%
Ciprofloxacin	75%
Amoxicillin	2%

The BNF recommends treating invasive diarrhoea (causing bloody diarrhoea and fever) with ciprofloxacin. Most viral or bacterial gastroenteritis do not require treatment. The BNF recommends antibiotics for bacterial gastroenteritis in severe infections or in immunocompromised patients. Clarithromycin is used for traveller's diarrhoea and non-invasive diarrhoeal illnesses when treatment is necessary.

Discuss (6) | Improve

Next question

Enteric fever (typhoid/paratyphoid)

The *Salmonella* group contains many members, most of which cause diarrhoeal diseases. They are aerobic, Gram-negative rods which are not normally present as commensals in the gut.

Typhoid and paratyphoid are caused by *Salmonella typhi* and *Salmonella paratyphi* (types A, B & C) respectively. They are often termed enteric fevers, producing systemic symptoms such as headache, fever, arthralgia.

Pathophysiology

- typhoid is transmitted via the faecal-oral route (also in contaminated food and water)

Features

- initially systemic upset as above
- relative bradycardia
- abdominal pain, distension
- constipation: although *Salmonella* is a recognised cause of diarrhoea, constipation is more common in typhoid
- rose spots: present on the trunk in 40% of patients, and are more common in paratyphoid

Possible complications include

- osteomyelitis (especially in sickle cell disease where *Salmonella* is one of the most common pathogens)
- GI bleed/perforation
- meningitis
- cholecystitis
- chronic carriage (1%, more likely if adult females)



Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

6 9

Typhoid fever review

Suggest link Report broken link

Media



Salmonella - a quick introduction and overview

Let's Learn About Bugs - YouTube 4 0



Salmonella

ATP - YouTube 7 1



Salmonella

SketchyMedical - YouTube 6 3

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Score: **22%**

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Back to top

A 40-year-old gentleman was admitted with fever, dry cough, headache, abdominal pain and diarrhoea. Around ten days previously he had been complaining of intermittent fevers and night sweats but otherwise felt well. In the last 3 days, he had developed generalised abdominal pain and watery diarrhoea, along with a dry cough and headache. Prior to this he had felt constipated only having opened his bowels once in three days which he thought was unusual as he had eaten a large amount of fruit off the market stools in South Korea where he had recently been on a business trip.

On examination, he was notably jaundiced, had a macular rash over his chest and had tender hepatomegaly. Observations revealed a temperature of 40.1ºC, heart rate 38/min, regular and a blood pressure of 130/90 mmHg. ECG showed a sinus bradycardia.

Blood tests revealed:

WBC	14.0 * 10^9/l
Neutrophils	12.0 * 10^9/l
CRP	230 mg/l
Bilirubin	52 µmol/l
ALP	80 u/l
ALT	200 u/l
Albumin	32 g/l

What investigation would you do to obtain the diagnosis?

- Blood cultures
- Abdominal ultrasound
- HIV test
- Lumbar puncture
- ECG

Submit answer

Reference ranges

Score: **24%**

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ALT	200 u/l
Albumin	32 g/l

What investigation would you do to obtain the diagnosis?

Blood cultures	66%
Abdominal ultrasound	18%
HIV test	11%
Lumbar puncture	3%
ECG	2%

Typhoid or enteric fever is the commonest serious tropical disease requiring treatment from Asia. Incubation period 7-18 days (range: 3-60 days). The highest incidence is found in south-central

Asia and south-east Asia.

Patients most commonly present with fever, headache, constipation/diarrhoea, malaise, anorexia, dry cough, abdominal pain, hepatosplenomegaly, Rose spot rash, bradycardia and potentially misleading symptoms including meningism may occur.

Transmission is usually from contaminated food or water, occasionally direct faecal-oral transmission, shellfish taken from sewage-polluted areas, ingestion of contaminated milk and milk products and flies may cause human infection through the transfer of infectious agents to foods. Around 25% of those who contract typhoid fever become chronic carriers, as bacteria persist in the biliary tract after symptoms have resolved.

Investigations may reveal raised WCC, deranged LFTS. Blood cultures have the highest yield within the first week of symptoms (sensitivity 40-80%). Diagnosis can also be made from stool and urine cultures (become positive after the first week) and bone marrow cultures.

Treatment is with quinolones most commonly ciprofloxacin. However, there has been increasing reports of quinolones resistance in patients returned from Asia and intravenous ceftriaxone may be used. If quinolone resistance is confirmed azithromycin or cefixime are suitable oral alternatives for uncomplicated disease. Treatment should be continued for 14 days to reduce the risk of relapse. The addition of steroids may be helpful in severe cases

Complications can include GI bleeding, intestinal or biliary perforation, acalculous cholecystitis, pneumonia, myocarditis, pancreatitis, UTIs, osteomyelitis, meningitis and typhoid encephalopathy,

Discuss (14) | Improve

Next question >

Enteric fever (typhoid/paratyphoid)

The *Salmonella* group contains many members, most of which cause diarrhoeal diseases. They are aerobic, Gram-negative rods which are not normally present as commensals in the gut.

Typhoid and paratyphoid are caused by *Salmonella typhi* and *Salmonella paratyphi* (types A, B & C) respectively. They are often termed enteric fevers, producing systemic symptoms such as headache, fever, arthralgia.

Pathophysiology

- typhoid is transmitted via the faecal-oral route (also in contaminated food and water)

Features

- initially systemic upset as above
- relative bradycardia
- abdominal pain, distension
- constipation: although *Salmonella* is a recognised cause of diarrhoea, constipation is more common in typhoid
- rose spots: present on the trunk in 40% of patients, and are more common in paratyphoid

Possible complications include

- osteomyelitis (especially in sickle cell disease where *Salmonella* is one of the most common pathogens)
- GI bleed/perforation
- meningitis
- cholecystitis
- chronic carriage (1%, more likely if adult females)



Next question

Textbooks

High-yield textbook

Extended textbook

Links

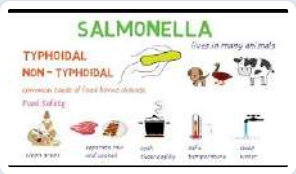
DermNet NZ

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Typhoid fever review

Suggest link Report broken link

Media



Salmonella - a quick introduction and overview

Let's Learn About Bugs - YouTube 4 0



Salmonella

ATP - YouTube 7 1



Salmonella

SketchyMedical - YouTube 6 3

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Score: **22%**

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A 33-year-old male with a previous history of asthma and HIV presents for review to HIV clinic. He has been noticing weight gain, marks on his abdomen and his partner has noticed that his face has been looking more heavy-set over the last two months. He was diagnosed as having HIV at the age of 19 following needle sharing and use of heroin. He has had good retroviral control following starting treatment at the age of 20 with tenofovir, emtricitabine, atazanavir and ritonavir. His asthma has been well controlled with only salbutamol until six months ago when due to recurrent exacerbation due to upper respiratory tract infection his treatment was escalated to include regular fluticasone. Repeat blood tests show an undetectable viral load and a CD4 count of 900 cells/microliter. What is the most likely cause of his symptoms?

- Weight gain secondary to tenofovir use
- Weight gain secondary to emtricitabine use
- Weight gain due to reduced exercise tolerance
- Endogenous Cushing's syndrome
- Iatrogenic Cushing's syndrome

Submit answer

Reference ranges

Score: **24%**

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Weight gain secondary to tenofovir use	14%
Weight gain secondary to emtricitabine use	14%
Weight gain due to reduced exercise tolerance	2%
Endogenous Cushing's syndrome	7%
Iatrogenic Cushing's syndrome	64%

The correct answer is iatrogenic Cushing's syndrome. HIV protease inhibitors are potent P450 inhibitors. This means that there are many possible interactions that should be checked before starting treatment. In this gentleman, hepatic enzyme inhibition has resulted in increased steroid dose from his regular inhaler for his asthma. This also explains the physical description showing standard Cushingoid features. Endogenous Cushing's syndrome is possible but is less likely, and weight gain from other causes would not explain all the physical changes described.

Source:
Saberi, Parya, Tony Phengrasamy, and Dong Phuong Nguyen. Inhaled Corticosteroid Use in HIV-Positive Individuals Taking Protease Inhibitors: A Review of Pharmacokinetics, Case Reports, and Clinical Management. HIV medicine 14.9 (2013): 519529. PMC. Web. 7 Mar. 2017.

HIV: management

Antiretroviral therapy (ART) involves a combination of at least three drugs, typically two nucleoside reverse transcriptase inhibitors (NRTI) and either a protease inhibitor (PI) or a non-nucleoside reverse transcriptase inhibitor (NNRTI). This combination both decreases viral replication but also reduces the risk of viral resistance emerging

Following the 2015 BHIVA guidelines it is now recommended that patients start ART as soon as they have been diagnosed with HIV, rather than waiting until a particular CD4 count, as was previously advocated.

Entry inhibitors

- maraviroc (binds to CCR5, preventing an interaction with gp41), enfuvirtide (binds to gp41, also known as a 'fusion inhibitor')
- prevent HIV-1 from entering and infecting immune cells

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), abacavir, emtricitabine, didanosine, lamivudine, stavudine, zalcitabine, tenofovir
- general NRTI side-effects: peripheral neuropathy
- tenofovir: used in BHIVAs two recommended regime NRTI. Adverse effects include renal impairment and ostesoporosis
- zidovudine: anaemia, myopathy, black nails
- didanosine: pancreatitis

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz
- side-effects: P450 enzyme interaction (nevirapine induces), rashes

Protease inhibitors (PI)

- examples: indinavir, nelfinavir, ritonavir, saquinavir
- side-effects: diabetes, hyperlipidaemia, buffalo hump, central obesity, P450 enzyme inhibition
- indinavir: renal stones, asymptomatic hyperbilirubinaemia
- ritonavir: a potent inhibitor of the P450 system

Integrase inhibitors

- block the action of integrase, a viral enzyme that inserts the viral genome into the DNA of the host cell
- examples: raltegravir, elvitegravir, dolutegravir

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

2016 HIV-1 infection guidelines

7 4

University of Liverpool

HIV Drug Interactions Checker

12 6

Suggest link

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Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube

1 0



Antiretroviral drugs

Medinaz - YouTube

38 2

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A returning traveller presents to the emergency department with a 10 day history of fever, cough and abdominal pain. He has spent the last 2 weeks in Jakarta, Indonesia. His vital signs are: temperature: 40.1ºC, heart rate 85 beats/minute, blood pressure 120/80 mmHg

On examination his spleen is enlarged and there is a rose spot rash over the chest. Blood culture grows salmonella typhi and the on-call doctor diagnoses enteric fever (typhoid).

If left untreated, what is the most important and serious complication that can occur within the following 2 weeks?

- Chronic carriage within gallbladder
- Bowel perforation and haemorrhage
- Splenic infarction and rupture
- Acute liver failure
- Bacterial meningitis

Submit answer

Reference ranges

Score: **24%**

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If left untreated, what is the most important and serious complication that can occur within the following 2 weeks?

Chronic carriage within gallbladder	5%
Bowel perforation and haemorrhage	69%
Splenic infarction and rupture	17%
Acute liver failure	4%
Bacterial meningitis	5%

The most serious and frequent complications of Typhoid are bowel perforation and haemorrhage. This is caused by bacterial destruction of the Peyer's patches in the small intestine and classically occurs late in the second week of illness or early in the third week. Other complications include myocarditis and endocarditis.

Chronic bacterial carriage in the gall bladder does occur, but this is not the most serious complication. It renders the patient chronically infective and capable of transmitting the disease. Acute liver failure does not occur, but mild jaundice may result from cholecystitis or hepatitis. Bacterial meningitis is a very rare complication not often reported.

Discuss (5) | Improve

Next question

Enteric fever (typhoid/paratyphoid)

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Features

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AI + Q 123

Next question >

Textbooks

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Links

DermNet NZ 6 9

Typhoid fever review

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Media



Salmonella - a quick introduction and overview

Let's Learn About Bugs - YouTube 4 0



Salmonella

ATP - YouTube 7 1



Salmonella

SketchyMedical - YouTube 6 3

Report broken media

Score: **22%**

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A 55 year-old patient presents with a 7 day history of nausea, breathlessness and feeling generally unwell. He is an HIV positive man who was recently started on anti-retroviral therapy in South Africa. On examination his chest is clear. His investigations are:

Chest X-ray: No abnormality seen

CD4 count	380 cells / mm³
Amylase	800 units / dL
Lactate	3.8 mmol / L

What is the most likely cause?

- ○ Pneumocystis jirovecii pneumonia (PCP) ×
- ○ Gallstone pancreatitis ×
- ○ Epstein-Barr Virus ×
- ○ Nucleoside reverse transcriptase inhibitor (NRTI) therapy ×
- ○ Non-nucleoside reverse transcriptase inhibitor (NNRTI) therapy ×

Submit answer

Reference ranges

Score: **24%**

1 ✓
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What is the most likely cause?

Pneumocystis jirovecii pneumonia (PCP)	10%
Gallstone pancreatitis	5%
Epstein-Barr Virus	3%
Nucleoside reverse transcriptase inhibitor (NRTI) therapy	60%
Non-nucleoside reverse transcriptase inhibitor (NNRTI) therapy	22%

NRTIs - in particular stavudine, didanosine and zidovudine - classically cause mitochondrial toxicity as an unwanted side effect. This can result in nausea, pancreatitis, lactic acidosis and lipoatrophy as demonstrated above.

Discuss (5) | Improve

Next question

HIV: management

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123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

7 4

2016 HIV-1 infection guidelines

University of Liverpool

12 6

HIV Drug Interactions Checker

Suggest link Report broken link

Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube

1 0



Antiretroviral drugs

Medinaz - YouTube

38 2

Report broken media

Score: **22%**

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A 25-year-old woman of Caucasian origin presents to the dermatology clinic for review. She has been volunteering in India for the last 12 months and has been living with a local family. She first noticed a small lesion on her lower leg around 6 weeks ago. It has since progressed significantly, so she has returned to the UK to seek medical attention. The lesion is painless. She has no other lesions elsewhere on the body. There is no past medical history of note but her brother has ulcerative colitis.

Her skin is examined, as shown below:



What is the most likely cause of this skin lesion?

- Buruli ulcer
- Cutaneous leishmaniasis
- Fish tank granuloma
- Pyoderma gangrenosum
- Yaws

Submit answer

Reference ranges

Score: **24%**

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Her skin is examined, as shown below:



What is the most likely cause of this skin lesion?

Buruli ulcer	3%
Cutaneous leishmaniasis	73%
Fish tank granuloma	2%
Pyoderma gangrenosum	19%
Yaws	2%

The image shows a large shallow ulcerating lesion on the lower leg, with a moist surface. It is on an exposed area of the body and is a solitary lesion. Combined with her travel history, the most likely cause is **cutaneous leishmaniasis** . Lesions in cutaneous leishmaniasis can also be dry or covered with a crust or scab. Leishmaniasis is transmitted by bites of the sandfly and is due to a number of species of the protozoan parasite *Leishmania* . The main species causing cutaneous leishmaniasis in Asia, Africa, the Mediterranean and the Middle East are *Leishmania major*, *Leishmania tropica* and *Leishmania aethiopica*. Cutaneous leishmaniasis in Central and South America is caused mainly by *Leishmania braziliensis* and *Leishmania mexicana*.

Buruli ulcer is incorrect. This is another tropical ulcerating disease caused by *Mycobacterium ulcerans* . However, most cases occur in sub-Saharan Africa and tropical parts of Australia, and it is rare in Asia. It is also less likely in this patient because it tends to affect children under the age of 15, and affects people living in poverty in rural areas. It is not commonly seen in travellers.

Fish tank granuloma is incorrect. This condition is caused by *Mycobacterium marinum* and can be seen in people who have had exposure to fish tanks or are involved in fish or shellfish preparation. However, unlike the lesion seen in this patient, skin lesions are generally plaque-like or nodular, rather than ulcerating. Lesions can occur on the legs but are more common on the hands and arms.

Pyoderma gangrenosum is incorrect. This diagnosis is less likely in this patient because the ulcers of pyoderma gangrenosum are generally very painful, and have a blue or purple undermined edge, neither of which are present in this case. The patient also has no personal history of an underlying condition associated with pyoderma gangrenosum.

Yaws is incorrect. This condition is one caused by *Treponema pallidum subspecies pertenue*. It is endemic in a small number of countries in Africa, Asia and the South Pacific, but is most common in Papua New Guinea. The primary stage presents with a papule that can later ulcerate. It is most common in children between 5 and 10. It is unlikely as a diagnosis in this patient because it is extremely unusual in returning travellers as it requires close skin contact (non-sexual contact) with open skin lesions for transmission. It has also been eradicated from India.

Discuss (1) Improve

Next question ›

Leishmaniasis

Leishmaniasis is caused by the intracellular protozoa *Leishmania*, which are spread by the bites of sandflies. Cutaneous, mucocutaneous leishmaniasis and visceral forms are seen

Cutaneous leishmaniasis

- caused by *Leishmania tropica* or *Leishmania mexicana*
- crusted lesion at the site of bite
- there may be an underlying ulcer
- it is typically diagnosed by doing a punch biopsy from the edge of the lesion allowing for both histology and culture
- cutaneous leishmaniasis acquired in South or Central America merits treatment due to the risk of mucocutaneous leishmaniasis whereas disease acquired in Africa or India can be managed more conservatively





Mucocutaneous leishmaniasis

- caused by *Leishmania braziliensis*
- skin lesions may spread to involve mucosae of nose, pharynx etc

Visceral leishmaniasis (kala-azar)

- mostly caused by *Leishmania donovani*
- occurs in the Mediterranean, Asia, South America, Africa
- fever, sweats, rigors
- massive splenomegaly. hepatomegaly
- poor appetite*, weight loss
    - occasionally patients may report increased appetite with paradoxical weight loss
- grey skin - 'kala-azar' means black sickness
- pancytopaenia secondary to hypersplenism
- the gold standard for diagnosis is bone marrow or splenic aspirate
- management
    - the BNF recommends sodium stibogluconate, an organic pentavalent antimony compound
    - amphotericin B may be used with or after treatment with an antimony compound

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians

2011 Leishmaniasis review

9 8

DermNet NZ

Leishmaniasis review

9 6

Suggest link

Report broken link

Media



What is Leishmaniasis? An introduction and overview

Let's Learn About Bugs - YouTube

5 2

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Score: **22%**

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A 34-year-old lady presents to the emergency department.

She moved to the UK from India 10 years ago but moved to the area 3 years ago. She works as a medical coder in the hospital.

She presents with a 3-day history of severe generalised headache, a stiff and painful neck, photophobia, and fever. Her husband reports she is drowsy and has been confused since that morning.

She is generally well and takes no medications. Her only previous medical history is of groin lymphadenopathy that was investigated 5 years ago. A biopsy was taken which showed a caseating granuloma. She had no further follow-up at that time.

Investigations:

Chest x-ray lung fields clear. No obvious abnormalities

Blood tests:

Haemoglobin	138 g/L
White cells	20.3x10^9/L
Platelets	310x10^9/L
CRP	>350mg/L
Glucose	6.8 mmol/L

Cerebrospinal Fluid (CSF): Appears turbid

Opening pressure	20 cmCSF
Protein	3280 mg/dL
White cells	466 cell/µL (polymorphonucleocytes 280 cell/µL and lymphocytes 206 cell/µL)
Red cells	4 cell/µL
Glucose	1.6 mmol/L
Lactate	5.6 mmol/L
CSF gram stain and PCR	pending

What is the best therapeutic regimen for this patient?

- Start empirical ceftriaxone, vancomycin and dexamethasone
- Await gram stain and viral PCR prior to starting a targeted therapy
- Start aciclovir, ceftriaxone and vancomycin
- Start isoniazid, rifampicin, pyrazinamide and ethambutol
- Start isoniazid, rifampicin, pyrazinamide and ethambutol as well as ceftriaxone, vancomycin and dexamethasone

Submit answer

Reference ranges

Score: **24%**

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A 34-year-old lady presents to the emergency department.

She moved to the UK from India 10 years ago but moved to the area 3 years ago. She works as a medical coder in the hospital.

She presents with a 3-day history of severe generalised headache, a stiff and painful neck, photophobia, and fever. Her husband reports she is drowsy and has been confused since that morning.

She is generally well and takes no medications. Her only previous medical history is of groin lymphadenopathy that was investigated 5 years ago. A biopsy was taken which showed a caseating granuloma. She had no further follow-up at that time.

Investigations:

Chest x-ray lung fields clear. No obvious abnormalities

Blood tests:

Haemoglobin	138 g/L
White cells	20.3x10^9/L
Platelets	310x10^9/L
CRP	>350mg/L
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Cerebrospinal Fluid (CSF): Appears turbid

Opening pressure	20 cmCSF
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White cells	466 cell/µL (polymorphonucleocytes 280 cell/µL and lymphocytes 206 cell/µL)
Red cells	4 cell/µL
Glucose	1.6 mmol/L
Lactate	5.6 mmol/L
CSF gram stain and PCR	pending

What is the best therapeutic regimen for this patient?

Start empirical ceftriaxone, vancomycin and dexamethasone	16%
Await gram stain and viral PCR prior to starting a targeted therapy	1%
Start aciclovir, ceftriaxone and vancomycin	5%
Start isoniazid, rifampicin, pyrazinamide and ethambutol	24%
Start isoniazid, rifampicin, pyrazinamide and ethambutol as well as ceftriaxone, vancomycin and dexamethasone	54%

This lady could have either bacterial meningitis or tuberculosis (TB) meningitis from her CSF results.

Although she has no chest signs of previous TB, she has had a previous biopsy suggesting she may have TB in her lymphatic system. This makes TB meningitis the most likely diagnosis, however, treatment should certainly be started empirically. This rules out answer 2 as the correct one. Since the gram stains and stain for acid fast bacilli would not yet be back from the lab, treatment should be started for both bacterial and TB meningitis.

Answers 1 and 3 do not include TB therapy and answer 4 does not cover for bacterial meningitis, therefore, these are incorrect answers.

https://www.britishinfection.org/files/3014/1617/4211/CNSTB2009Thwaites.pdf

Discuss (12) Improve

Next question

Tuberculosis: management

The standard therapy for treating **active tuberculosis** is:

Initial phase - first 2 months (RIPE)

- Rifampicin
- Isoniazid
- Pyrazinamide

- Ethambutol (the 2006 NICE guidelines now recommend giving a 'fourth drug' such as ethambutol routinely - previously this was only added if drug-resistant tuberculosis was suspected)

Continuation phase - next 4 months

- Rifampicin
- Isoniazid

The treatment for **latent tuberculosis** is 3 months of isoniazid (with pyridoxine) and rifampicin OR 6 months of isoniazid (with pyridoxine)

Patients with **meningeal tuberculosis** are treated for a prolonged period (at least 12 months) with the addition of steroids

Directly observed therapy with a three times a week dosing regimen may be indicated in certain groups, including:

- homeless people with active tuberculosis
- patients who are likely to have poor concordance
- all prisoners with active or latent tuberculosis

Complications of treatment

Immune reconstitution disease

- occurs typically 3-6 weeks after starting treatment
- often presents with enlarging lymph nodes

Drug adverse effects

- rifampicin
  - potent liver enzyme inducer
  - hepatitis, orange secretions
  - flu-like symptoms
- isoniazid
  - peripheral neuropathy: prevent with pyridoxine (Vitamin B6)
  - hepatitis, agranulocytosis
  - liver enzyme inhibitor
- pyrazinamide
  - hyperuricaemia causing gout
  - arthralgia, myalgia
  - hepatitis
- ethambutol
  - optic neuritis: check visual acuity before and during treatment

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Cochrane 7 7

Corticosteroids for managing tuberculous meningitis

NICE 7 6

2016 Tuberculosis guidelines

Clinical Knowledge Summaries 8 3

Tuberculosis

Suggest link Report broken link

Media



Tuberculosis

Osmosis - YouTube 2 0

Report broken media

Score: **22%**

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A 54-year-old man presents to the emergency department with one day of fever and a severely painful left leg. He has a past medical history of hypertension. He takes ramipril. He smokes cannabis occasionally and drinks approximately 10 units of alcohol a week. He has recently completed treatment with aciclovir for an episode of chicken-pox.

His observations are heart rate 111 beats per minute, blood pressure 86/48 mmHg, respiratory rate 21/minute, oxygen saturations 97% on room air and temperature 38.4ºC.

On examination, his left thigh is erythematous, tense and tender to touch. There is palpable crepitus.

Blood tests:

Hb	108 g/L	Male: (135-180) Female: (115 - 160)
Platelets	555 * 10^9/L	(150 - 400)
WBC	23.1 * 10^9/L	(4.0 - 11.0)
Na^+	131 mmol/L	(135 - 145)
K^+	4.8 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	191 µmol/L	(55 - 120)
CRP	351 mg/L	(< 5)

Given the likely diagnosis, what is his most significant risk factor the development of this condition?

- Acute renal failure
- Cannabis use
- Chickenpox infection
- History of hypertension
- Weekly alcohol intake

Submit answer

Reference ranges

Score: **24%**

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Creatinine	191 µmol/L	(55 - 120)
CRP	351 mg/L	(< 5)

Given the likely diagnosis, what is his most significant risk factor the development of this condition?

- Acute renal failure — 8%
- Cannabis use — 9%
- Chickenpox infection — 75%
- History of hypertension — 2%
- Weekly alcohol intake — 6%

Chickenpox is a risk factor for invasive group A streptococcal soft tissue infections including necrotizing fasciitis

Important for me Less important

Chickenpox infection is correct. The patient has necrotizing fasciitis as evidenced by clinical evidence of soft tissue infection, with severe pain and crepitus. Preceding chickenpox infection is a risk factor for this condition.

Acute renal failure is incorrect. Chronic renal failure is associated with the development of necrotizing fasciitis. In this case, he has developed an acute kidney injury in the context of septic shock. Renal failure is a consequence of necrotizing fasciitis rather than a cause in this case.

Cannabis use is incorrect. Intravenous drug abuse is a risk factor for necrotizing fasciitis but not inhaled cannabis use.

History of hypertension is incorrect. Necrotizing fasciitis is associated with chronic disease. However, the conditions commonly implicated are diabetes and peripheral vascular disease rather than hypertension.

Weekly alcohol intake is incorrect. Alcohol abuse is associated withe the development of necrotizing fasciitis but he drinks within normal limits (i.e. < 14 units/week).

Discuss (1) Improve

Next question

Chickenpox

Chickenpox is caused by primary infection with varicella zoster virus. Shingles is a reactivation of the dormant virus in dorsal root ganglion

Chickenpox is highly infectious

- spread via the respiratory route
- can be caught from someone with shingles
- infectivity = 4 days before rash, until 5 days after the rash first appeared*
- incubation period = 10-21 days

Clinical features (tend to be more severe in older children/adults)

- fever initially

- itchy, rash starting on head/trunk before spreading. Initially macular then papular then vesicular
- systemic upset is usually mild

Management is supportive

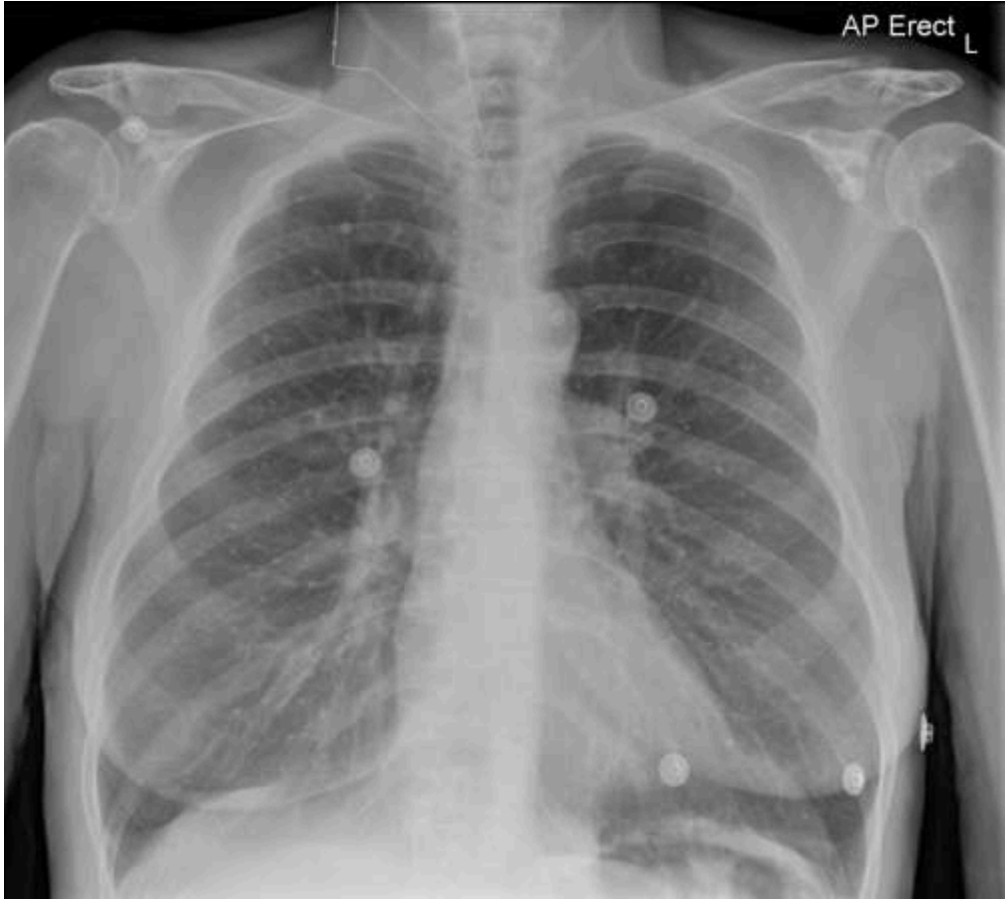
- keep cool, trim nails
- calamine lotion
- school exclusion: NICE state the following: *Advise that the most infectious period is 1-2 days before the rash appears, but infectivity continues until all the lesions are dry and have crusted over (usually about 5 days after the onset of the rash).*
- immunocompromised patients and newborns with peripartum exposure should receive varicella zoster immunoglobulin (VZIG). If chickenpox develops then IV aciclovir should be considered

A common complication is secondary bacterial infection of the lesions

- NSAIDs may increase this risk
- whilst this commonly may manifest as a single infected lesion/small area of cellulitis, in a small number of patients invasive group A streptococcal soft tissue infections may occur resulting in necrotizing fasciitis

Rare complications include

- pneumonia
- encephalitis (cerebellar involvement may be seen)
- disseminated haemorrhagic chickenpox
- arthritis, nephritis and pancreatitis may very rarely be seen



© Image used on license from Radiopaedia

Chest x-ray showing miliary opacities secondary to healed varicella pneumonia. Multiple tiny calcific miliary opacities noted throughout both lungs. These are of uniform size and dense suggesting calcification. There is no focal lung parenchymal mass or cavitating lesion seen. The appearances are characteristic for healed varicella pneumonia.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Greenbook 8 6

Varicella

Public Health England 9 6

Chapter 9: managing specific infectious diseases

Suggest link Report broken link

Media



Varicella zoster virus

Osmosis - YouTube 6 0

Report broken media

Score: **22%**

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Back to top

A 35-year-old man presents to the emergency department with three days of headache, fever, diarrhoea and malaise. He has recently returned from Angola, where he was taking pictures of neolithic wall paintings. He has no past medical history and is on no regular medications. He works as a photo-journalist for a major international publication.

His observations are heart rate 124 beats per minute, respiratory rate 21/minute, blood pressure 85/54 mmHg, oxygen saturations 96% on room and air and temperature 39.3ºC.

Clinical examination reveals an unwell man. His capillary refill time is four seconds and his skin turgor is increased. He is oozing blood from his mucus membranes. Chest auscultation is normal. Heart sounds are normal with no murmurs. His abdomen is soft, diffusely tender but not peritonitic.

Intravenous access is obtained and intravenous fluids and empirical broad-spectrum antibiotics are administered. It is noted that he is oozing blood from his cannulae sites.

Blood tests:

Hb	111 g/L	Male: (135-180) Female: (115 - 160)
Platelets	543 * 10^9/L	(150 - 400)
WBC	5.2 * 10^9/L	(4.0 - 11.0)
Na^+	129 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	14.1 mmol/L	(2.0 - 7.0)
Creatinine	156 µmol/L	(55 - 120)
CRP	80 mg/L	(< 5)
Bilirubin	28 µmol/L	(3 - 17)
ALP	111 u/L	(30 - 100)
ALT	143 u/L	(3 - 40)
Î³GT	99 u/L	(8 - 60)
Albumin	28 g/L	(35 - 50)

Based on the probable diagnosis, to which zoonotic vector has he likely been exposed?

- Bats
- Birds
- Cattle
- Pigs
- Rats

Submit answer

Reference ranges

Score: **24%**

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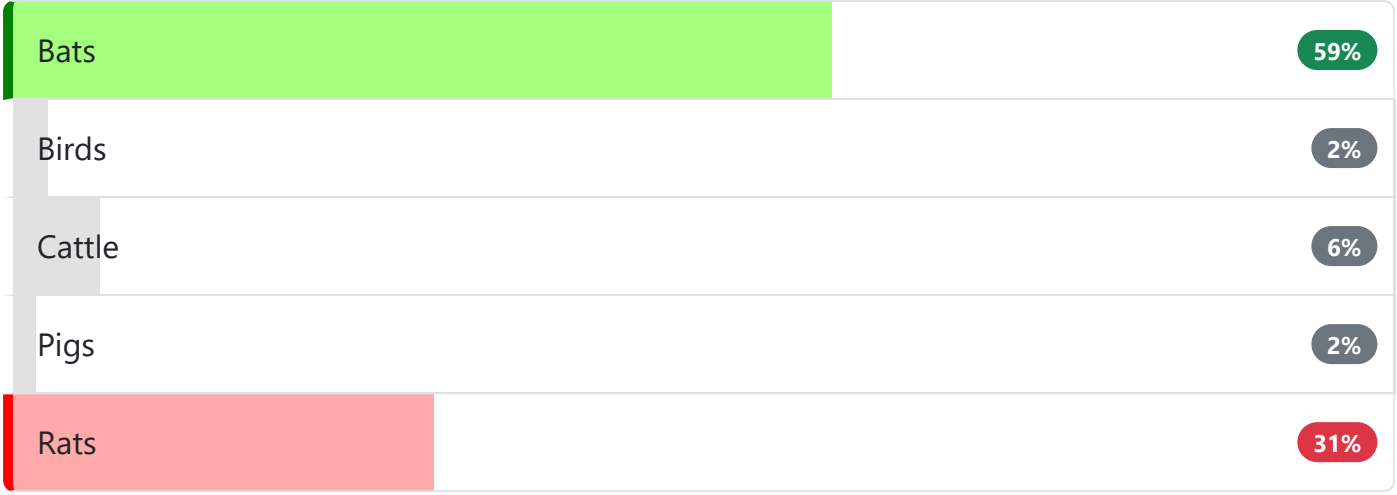
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Î³GT	99 u/L	(8 - 60)
Albumin	28 g/L	(35 - 50)

Based on the probable diagnosis, to which zoonotic vector has he likely been exposed?



Exposure to bats/caves is a risk factor for Marburg virus, which results in a viral haemorrhagic fever

Important for me Less important

Bats are the correct answer. This man has evidence of viral haemorrhagic fever (VHF) with hypotension and multi-organ dysfunction. He has returned from a region where Marburg virus has previously been found. Additionally, he has been taking photos of neolithic wall paintings, which are typically found in caves. Exposure to bats and caves are risk factors for Marburg virus.

Birds are not the correct answer. While there is some suggestion that birds can carry infected ticks from continent to continent and thus spread viruses causing (VHF), this is not a common vector for the animal to human transmission of these diseases.

Cattle is incorrect. While people can occasionally contract Crimean-Congo haemorrhagic fever from exposure to infected cattle, there has never been an outbreak of this condition in Angola and he has not been exposed to slaughtered animals.

Pigs are incorrect. This is not a common zoonotic vector for transmission of viral haemorrhagic fever.

Rats are incorrect. While exposure to rats can result in hantavirus and haemorrhagic fever and renal dysfunction, this is more common in Asia, the Americas and Europe, rather than Africa.

Discuss (8) Improve

Next question

Viral haemorrhagic fevers

Viral haemorrhagic fevers (VHFs) are a group of viral infections that result in presentations ranging from a flu-like illness to multisystem failure.

Examples of viral haemorrhagic fever (VHF) include:

- Flaviviridae: dengue, yellow fever
- Arenaviridae: Lassa fever
- Filoviridae: Ebola virus, Marburg virus
- Bunyaviridae: Hantaviruses, Crimean-Congo haemorrhagic fever, Rift Valley fever

Features

- flu-like symptoms
- abdominal pain
- haemorrhage
  - petechiae, bruising
  - bloody diarrhoea, haematemesis, haemoptysis
  - disseminated intravascular coagulation
- multiorgan failure

Prevention

- a yellow fever vaccine is available
- a dengue and Ebola vaccine have been developed but are now widely used at the current time
- no vaccines are available for the other VHFs

Management

- treatment is generally supportive
- ribavirin is used to treat Lassa fever

The more common VHFs such as dengue, Ebola and yellow fever are covered in more detail elsewhere in the notes.

Lassa fever

Lassa fever is contracted by contact with the excreta of infected African rats (Mastomys rodent) or by person-to-person spread.

Marburg virus

Causes a VHF very similar to Ebola.

Risk factors for transmissions include caves/bats and primates.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

HPA

2 1

Management of viral haemorrhagic fever

Suggest link

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Score: **22%**

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Back to top

A 43 year old man attends for follow up at the Infectious Diseases clinic. He is an HIV-positive patient who has been well while on treatment for the last 13 years. His current treatment regimen is emtricitabine, atazanavir and tenofovir. He has however begun to notice altered taste and a difficulty in swallowing over the last couple of months. In the last few days he has even noticed a white coating in his mouth that is not removed by vigorous scrubbing with his toothbrush. He describes being compliant with his medications and not taking any illicit drugs. He no longer has unprotected sexual intercourse. The only change that is identified is that he recently visited his GP with non-specific symptoms and was started on a new medication.

CD4 count in clinic 4 months ago 526
CD4 count currently 210

What medication is the GP likely to have started?

- Lansoprazole
- Aspirin
- Amitriptyline
- Paracetamol
- Simvastatin

Submit answer

Reference ranges

Score: **24%**

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CD4 count in clinic 4 months ago 526
CD4 count currently 210

What medication is the GP likely to have started?

Lansoprazole	61%
Aspirin	4%
Amitriptyline	15%
Paracetamol	1%
Simvastatin	19%

Interactions between anti-reteroviral drugs and other more common medications are many and varied. It is a complex area but is one that is becoming more prominent given the larger prevalance of HIV. HIV is no longer a death sentence and with current treatments patients can expect to have an almost normal life expectancy. Full knowledge of all the possible interactions is unrealistic but some of the main ones should be kept in mind when prescribing any new medication. There is an excellent resource kept by the University of Liverpool that can be found at https://www.hiv-druginteractions.org/

In the case detailed above the patient is likely to have been started on a PPI such as Lansoprazole. Lansoprazole is known to dangerously lower the efficacy of atazanavir and this is exhibited by the dramatic fall in CD4 count. This patient also seems to have developed oesophageal candidiasis and so urgent management to manage his HIV is required to avoid potentially deadly opportunistic infections.

HIV: management

Antiretroviral therapy (ART) involves a combination of at least three drugs, typically two nucleoside reverse transcriptase inhibitors (NRTI) and either a protease inhibitor (PI) or a non-nucleoside reverse transcriptase inhibitor (NNRTI). This combination both decreases viral replication but also reduces the risk of viral resistance emerging

Following the 2015 BHIVA guidelines it is now recommended that patients start ART as soon as they have been diagnosed with HIV, rather than waiting until a particular CD4 count, as was previously advocated.

Entry inhibitors

- maraviroc (binds to CCR5, preventing an interaction with gp41), enfuvirtide (binds to gp41, also known as a 'fusion inhibitor')
- prevent HIV-1 from entering and infecting immune cells

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), abacavir, emtricitabine, didanosine, lamivudine, stavudine, zalcitabine, tenofovir
- general NRTI side-effects: peripheral neuropathy
- tenofovir: used in BHIVAs two recommended regime NRTI. Adverse effects include renal impairment and ostesoporosis
- zidovudine: anaemia, myopathy, black nails
- didanosine: pancreatitis

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz
- side-effects: P450 enzyme interaction (nevirapine induces), rashes

Protease inhibitors (PI)

- examples: indinavir, nelfinavir, ritonavir, saquinavir
- side-effects: diabetes, hyperlipidaemia, buffalo hump, central obesity, P450 enzyme inhibition
- indinavir: renal stones, asymptomatic hyperbilirubinaemia
- ritonavir: a potent inhibitor of the P450 system

Integrase inhibitors

- block the action of integrase, a viral enzyme that inserts the viral genome into the DNA of the host cell
- examples: raltegravir, elvitegravir, dolutegravir

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Next question

Textbooks

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7 4

2016 HIV-1 infection guidelines

University of Liverpool

12 6

HIV Drug Interactions Checker

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Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube 1 0



Antiretroviral drugs

Medinaz - YouTube 38 2

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A 48-year-old woman originally from South Africa has lived in the UK for 17 years and works as a librarian. She was diagnosed with HIV 18 months ago after she discovered her husband had been having an affair. Her lowest CD4 count was 211 /mm3. She was commenced on Anti-Retroviral Therapy (ART) on diagnosis with Tenofovir, Emtricitabine and Kaletra (Lopinavir/Ritonavir) and Co-trimoxazole.

Her past medical history prior to diagnosis was unremarkable. She has been well on her ART until 3 weeks ago when she was seen in HIV clinic with a 4-week history of a cough and weight loss of 3 kilogrammes.

She had a chest x-ray showing new right apical nodular opacities and had scanty Acid Fast Bacilli on sputum smear microscopy. Her CD4 count was 312 /mm3 with and she was commenced on standard TB therapy with Rifampicin, Isoniazid, Ethambutol and Pyrazinamide, as well as switching Kaletra to Efavirenz.

Today she has been brought into hospital by the Police, she was found naked wandering in her street, accosting passers-by and asking them to help her remove a device planted under her skin by the CIA to monitor her thoughts.

Her observations are within normal limits. She is moderately difficult to examine but there is no focal neurology detectable. She has mildly reduced air entry in her right upper lobe and her examination is otherwise normal.

Results:

Hb	8.1 g/dl
MCV	84 fl
Platelets	86 * 10^9/l
WBC	4.5 * 10^9/l

CRP	27 mg/l
ESR	41 mm/hr

Urea & electrolytes, liver function tests and thyroid function tests were normal. A CT head and lumbar puncture were also unremarkable.

What is the cause of her admission?

- TB meningitis
- HIV encephalopathy
- Ethambutol toxicity
- Efavirenz toxicity
- Immune thrombocytopaenic purpura

Submit answer

Reference ranges

Score: **24%**

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Urea & electrolytes, liver function tests and thyroid function tests were normal. A CT head and lumbar puncture were also unremarkable.

What is the cause of her admission?

TB meningitis	5%
HIV encephalopathy	21%
Ethambutol toxicity	7%
Efavirenz toxicity	64%
Immune thrombocytopaenic purpura	3%

This is Efavirenz toxicity.

Rifampicin causes potent enzyme induction and therefore dramatically reduced the level of Protease Inhibitors. Efavirenz is less susceptible to inducing effects Rifampicin, therefore the switch to Efavirenz was logical.

Efavirenz however can cause neuropsychiatric toxicity (due to variable metabolism which is genetically determined) which is the cause of psychosis here. Normal LP excludes TB meningitis. Ethambutol causes optic neuritis, so monitoring of visual acuity during treatment is essential. HIV encephalopathy is possible but unlikely to present so acutely and the temporal relation to Efavirenz introduction, in this case, is the key.

Discuss (7) Improve

Next question

HIV: management

Antiretroviral therapy (ART) involves a combination of at least three drugs, typically two nucleoside reverse transcriptase inhibitors (NRTI) and either a protease inhibitor (PI) or a non-nucleoside reverse transcriptase inhibitor (NNRTI). This combination both decreases viral replication but also reduces the risk of viral resistance emerging

Following the 2015 BHIVA guidelines it is now recommended that patients start ART as soon as they have been diagnosed with HIV, rather than waiting until a particular CD4 count, as was previously advocated.

Entry inhibitors

- maraviroc (binds to CCR5, preventing an interaction with gp41), enfuvirtide (binds to gp41, also known as a 'fusion inhibitor')
- prevent HIV-1 from entering and infecting immune cells

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), abacavir, emtricitabine, didanosine, lamivudine, stavudine, zalcitabine, tenofovir
- general NRTI side-effects: peripheral neuropathy
- tenofovir: used in BHIVAs two recommended regime NRTI. Adverse effects include renal impairment and ostesoporosis
- zidovudine: anaemia, myopathy, black nails
- didanosine: pancreatitis

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

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Antiretroviral drugs

Medinaz - YouTube

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A 26-year-old woman presents to the emergency department with right upper quadrant pain which is worse when she takes a deep breath in. Her symptoms started three days earlier and have been progressively getting worse. She has no past medical history and takes no medications apart from the oral contraceptive pill and paracetamol to help control the pain. She has also noticed an increase in vaginal discharge over the last two weeks and has noticed an unpleasant smell with it. She works in an investment bank. On examination, she has right upper quadrant tenderness, but the abdomen is soft and no organomegaly is noticed. Abdominal ultrasound demonstrates the presence of gallstones in the gallbladder but is otherwise normal. What treatment is most likely to resolve the cause of her underlying problem?

- Cholecystectomy
- Trimethoprim
- Antiviral treatment
- Antiretroviral treatment
- Ceftriaxone

Submit answer

Reference ranges

Score: **24%**

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Cholecystectomy	14%
Trimethoprim	8%
Antiviral treatment	4%
Antiretroviral treatment	5%
Ceftriaxone	69%

Intramuscular ceftriaxone is the treatment of choice for *Gonorrhoea*

Important for me Less important

The correct answer is ceftriaxone. This woman has unfortunately developed Fitz-Hugh-Curtis syndrome following pelvic inflammatory diseases. The key aspects are RUQ pain worse with breathing; symptomatic of peri-hepatic adhesions. This combined with vaginal discharge are highly suggestive of Fitz-Hugh-Curtis syndrome, which can be treated with antibiotics. Cholecystectomy would be appropriate management if the US had found evidence of cholecystitis. Trimethoprim would be appropriate if there was evidence of urinary symptoms, whilst there is no evidence to suggest that this is an AIDS-defining illness needing antiretroviral treatment. Antiviral treatment would imply viral hepatitis, but there is no clear evidence of hepatitis.

Gonorrhoea

Gonorrhoea is caused by the Gram-negative diplococcus *Neisseria gonorrhoeae*. Acute infection can occur on any mucous membrane surface, typically genitourinary but also rectum and pharynx. The incubation period of gonorrhoea is 2-5 days

Features

- males: urethral discharge, dysuria
- females: cervicitis e.g. leading to vaginal discharge
- rectal and pharyngeal infection is usually asymptomatic

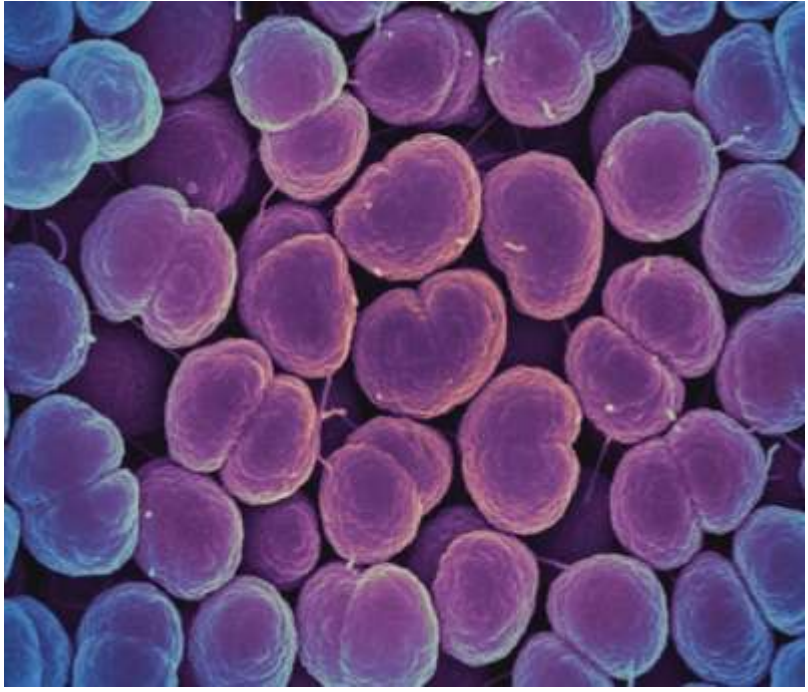
Microbiology

- immunisation is not possible and reinfection is common due to antigen variation of type IV pili (proteins which adhere to surfaces) and Opa proteins (surface proteins which bind to receptors on immune cells)

Local complications that may develop include urethral strictures, epididymitis and salpingitis (hence may lead to infertility). Disseminated infection may occur - see below

Management

- ciprofloxacin used to be the treatment of choice. However, there is increased resistance to ciprofloxacin (around 36% in the UK) and therefore cephalosporins are now more widely used
- there was a change in the 2019 British Society for Sexual Health and HIV (BASHH) guidelines. Previously the first-line treatment was IM ceftriaxone + oral azithromycin. The new first-line treatment is a single dose of IM ceftriaxone 1g (i.e. no longer add azithromycin). If sensitivities are known (and the organism is sensitive to ciprofloxacin) then a single dose of oral ciprofloxacin 500mg should be given
- if ceftriaxone is refused (e.g. needle-phobic) then oral cefixime 400mg (single dose) + oral azithromycin 2g (single dose) should be used



Colorized scanning electron micrograph of *Neisseria gonorrhoeae*. Credit: NIAID

Disseminated gonococcal infection (DGI) and gonococcal arthritis may also occur, with gonococcal infection being the most common cause of septic arthritis in young adults. The pathophysiology of DGI is not fully understood but is thought to be due to haematogenous spread from mucosal infection (e.g. Asymptomatic genital infection). Initially there may be a classic triad of symptoms: tenosynovitis, migratory polyarthritis and dermatitis. Later complications include septic arthritis, endocarditis and perihepatitis (Fitz-Hugh-Curtis syndrome)

Key features of disseminated gonococcal infection

- tenosynovitis
- migratory polyarthritis
- dermatitis (lesions can be maculopapular or vesicular)

Next question

Textbooks

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Links

British Society for Sexual Health and HIV (BASHH) 11 6

2019 Gonorrhoea guidelines

Clinical Knowledge Summaries 10 3

Gonorrhoea guidelines

Suggest link Report broken link

Media



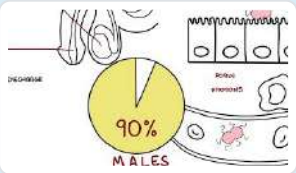
Neisseria gonorrhoeae

Osmosis - YouTube 2 0



Neisseria gonorrhoeae

ATP - YouTube 1 0



Neisseria gonorrhea (STI) symptoms, pathophysiology, complications and treatment

Armando Hasudungan - YouTube 0 0

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A 28-year-old female presents with a 4-day history of fevers and joint pain. She has recently returned from a 3 month gap year trip to the South-East Asia three days ago and reports no ill health during her travels. She has no past medical history, does not smoke, drinks minimal alcohol and denies the use of illicit drugs. During her travels, she reports two episodes of unprotected sexual contact with a non-regular partner. Although she knew she would be entering a malaria area and was indeed bitten by mosquitoes on a number of occasions, she did not take any malaria prophylaxis.

On examination, heart sounds and chest examination are both normal. A maculopapular rash is noted on her left thigh and right upper arm, with bilateral conjunctival injection. Abdominal examination reveals a soft abdomen with no masses. She has a significantly joint and muscle pains, limiting your neurological examination. She is alert and orientated to time and place, scoring 10/10 on abbreviated mental testing. Her blood tests are as follows:

Hb	109 g/l
Platelets	45 * 10^9/l
WBC	3.5 * 10^9/l

Na^+	137 mmol/l
K^+	3.8 mmol/l
Urea	7.1 mmol/l
Creatinine	100 µmol/l
CRP	70 mg/l

Bilirubin	7 µmol/l
ALP	30 u/l
ALT	162 u/l

Her first malaria film is negative and a chest radiograph is unremarkable.

What is the most likely diagnosis?

- Gonorrhoea with reactive arthritis
- HIV seroconversion
- Dengue fever

Malaria falciparum

Typhoid

Submit answer

Reference ranges

Score: **22%**

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Her first malaria film is negative and a chest radiograph is unremarkable.

What is the most likely diagnosis?

Gonorrhoea with reactive arthritis	13%
HIV seroconversion	13%
Dengue fever	65%

Malaria falciparum 5%

Typhoid 4%

Retro-orbital headache, fever, facial flushing, rash, thrombocytopenia in returning traveller → ?dengue

Important for me Less important

The context of this patient should alert you to that of fever in a returning traveller. This is a patient who presents with a combination of fever, myalgia and arthralgia, skin rash, conjunctival injection, raised ALT, thrombocytopaenia and leucopenia, strongly suggestive of dengue fever. The key differential for a returning traveller from South East Asia, where ingestion of contaminated water is possible, is to consider is also typhoid fever, which also presents with a skin rash (classically a rose spot) with diarrhoea and vomiting much more prominent. Although she had unprotected sex during her travel, gonorrhoea presents more typically with a migratory arthritis and tenosynovitis with discharge. Leucopenia is classical of dengue fever and does not necessarily suggest immunodeficiency, possibly secondary to HIV.

Discuss (8) Improve

Next question

Dengue fever

Dengue fever is a viral infection that can progress to viral haemorrhagic fever (other examples include yellow fever, Lassa fever, Ebola).

Aetiology

- dengue virus is a RNA virus of the genus Flavivirus
- transmitted by the *Aedes aegypti* mosquito
- incubation period of 7 days

Clinical features

Patients with dengue fever can be classified as follows:

- dengue fever:
  - without warning signs
  - with warning signs

- severe dengue (dengue haemorrhagic fever)

Dengue fever

- fever
- headache (often retro-orbital)
- myalgia, bone pain and arthralgia ('break-bone fever')
- pleuritic pain
- facial flushing (dengue)
- maculopapular rash
- haemorrhagic manifestations e.g. positive tourniquet test, petechiae, purpura/ecchymosis, epistaxis
- 'warning signs' include:
  - abdominal pain
  - hepatomegaly
  - persistent vomiting
  - clinical fluid accumulation (ascites, pleural effusion)

Severe dengue (dengue haemorrhagic fever)

- this is a form of disseminated intravascular coagulation (DIC) resulting in:
  - thrombocytopenia
  - spontaneous bleeding
- around 20-30% of these patients go on to develop dengue shock syndrome (DSS)

Investigations

- typically blood results
  - leukopenia, thrombocytopenia, raised aminotransferases
- diagnostic tests
  - serology
  - nucleic acid amplification tests for viral RNA
  - NS1 antigen test

Treatment

- entirely symptomatic e.g. fluid resuscitation, blood transfusion etc
- no antivirals are currently available

123

Next question

Textbooks

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Extended textbook

Links

Royal College of Physicians

8 14

2011 Dengue fever review

Postgraduate Medical Journal

6 9

Review of dengue fever

Suggest link

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A 39-year-old pet shop owner is referred to the dermatology clinic due to lesions on his right hand. He describes noticing a lump over his 2nd metacarpophalangeal joint 2 months ago, this has since increased in size and crusted over, and further similar lesions have appeared. He denies pain or itching and has tried a number of topical emollients without improvement. He has no past medical history of note and no allergies.

On examination, there are multiple crusted lesions measuring 1-2cm in diameter on his knuckles and the dorsum of his hand. There are no other skin or mucosal changes.

A biopsy is taken for analysis:

Skin biopsy	Granulomatous dermatitis with inflammatory infiltrate

What is the most likely causative organism?

- *Human papilloma virus*
- *Mycobacterium leprae*
- *Mycobacterium marinum*
- *Mycobacterium tuberculosis*
- *Pseudomonas aeruginosa*

Submit answer

Reference ranges

Score: **22%**

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Back to top

A 39-year-old pet shop owner is referred to the dermatology clinic due to lesions on his right hand. He describes noticing a lump over his 2nd metacarpophalangeal joint 2 months ago, this has since increased in size and crusted over, and further similar lesions have appeared. He denies pain or itching and has tried a number of topical emollients without improvement. He has no past medical history of note and no allergies.

On examination, there are multiple crusted lesions measuring 1-2cm in diameter on his knuckles and the dorsum of his hand. There are no other skin or mucosal changes.

A biopsy is taken for analysis:

Skin biopsy	Granulomatous dermatitis with inflammatory infiltrate

What is the most likely causative organism?

Human papilloma virus	7%
Mycobacterium leprae	21%
Mycobacterium marinum	70%
Mycobacterium tuberculosis	1%
Pseudomonas aeruginosa	1%

Fish tank granuloma is caused by *Mycobacterium marinum*

Important for me Less important

AI

This patient has an infection with the bacterium ***Mycobacterium marinum***, a species of mycobacteria commonly contracted through exposure to swimming pool water or contact with fish e.g. through fish tanks. This patient is a pet shop owner and has likely contracted the illness through occupational exposure. Notably, in such patients, the dominant hand is most frequently affected. After a 3-4 week incubation period, a single lump or pustule appears that breaks down to form a crusted lesion. The knuckles are commonly affected and a biopsy of the lesion demonstrates granulomatous dermatitis and inflammatory infiltrate within the dermis. Treatment is with antibiotics e.g. doxycycline.

Human papilloma virus (HPV) is a common cause of viral warts that are commonly located on the cutaneous surface (e.g. hands, knees and feet) or genitalia. Common warts found on the hands (also known as verruca vulgaris) are hyperkeratotic, rough and papillomatous lesions. However, on dermoscopy, multiple papillary capillaries can be visualised rather than granulomatous dermatitis with inflammatory infiltrate.

Mycobacterium leprae causes leprosy. Symptoms of leprosy include hypopigmented patches of skin associated with sensory loss. The areas of skin affected by the disease are not limited by exposure, in contrast to this patient's presentation.

Mycobacterium tuberculosis can cause cutaneous symptoms such as tuberculous gumma from the haematogenous spread of disease, lupus vulgaris and miliary tuberculosis where there are multiple millet-sized red skin lesions. These lesions are not seen in this patient and there is no other history to suggest underlying tuberculosis.

Pseudomonas aeruginosa is implicated in skin infections such as external otitis or hot tub folliculitis (acquired from chlorinated hot tubs). Infection is particularly more common in the immunocompromised and is unlikely to be the causative pathogen in this case.

Discuss | Improve

Next question ›

Mycobacterium marinum

Mycobacterium marinum is one of many mycobacteria that can cause disease in humans. Fish tank granuloma typically presents in patients who have had an exposure to, or frequently work with fish. It has an incubation period of 3-4 weeks and lesions can be painful or painless. A cut or break in the skin can be enough for the organism to enter the blood stream and track up the lymphatic system (sporotrichoid spread). Treatment options include tetracyclines, fluoroquinolones, sulfonamides and macrolides.

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

4 0

Atypical mycobacterial infection

Suggest link

Report broken link

Score: **16.8%**

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Back to top

A 65-year-old man has just returned from a trip to India one week ago. He has had bloody diarrhoea and fevers for the last two weeks and noted rose coloured spots on his abdomen yesterday. Apart from a prosthetic aortic valve, he has no significant past medical history. His blood tests indicate raised inflammatory markers and stool microbiology has found a gram-negative bacillus identified as a non-typhoidal *Salmonella*. Sensitivities are pending. Which one of the following options is best initial empiric management?

- Amoxicillin + clavulanic acid
- Gentamicin
- Ciprofloxacin
- Clindamycin
- Not for antibiotics

Submit answer

Reference ranges

Score: **22%**

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Amoxicillin + clavulanic acid	3%
Gentamicin	3%
Ciprofloxacin	85%
Clindamycin	3%
Not for antibiotics	5%

AI

According to the NICE guidelines, anyone above the age of 50, immunocompromised or has cardiac valve disease/endovascular abnormalities should be treating empirically with ciprofloxacin 500mg BD when they have been diagnosed with non-typhoidal *Salmonella* gastroenteritis.

Discuss (4) | Improve

Next question >

Enteric fever (typhoid/paratyphoid)

The *Salmonella* group contains many members, most of which cause diarrhoeal diseases. They are aerobic, Gram-negative rods which are not normally present as commensals in the gut.

Typhoid and paratyphoid are caused by *Salmonella typhi* and *Salmonella paratyphi* (types A, B & C) respectively. They are often termed enteric fevers, producing systemic symptoms such as headache, fever, arthralgia.

Pathophysiology

- typhoid is transmitted via the faecal-oral route (also in contaminated food and water)

Features

- initially systemic upset as above
- relative bradycardia
- abdominal pain, distension
- constipation: although *Salmonella* is a recognised cause of diarrhoea, constipation is more common in typhoid
- rose spots: present on the trunk in 40% of patients, and are more common in paratyphoid

Possible complications include

- osteomyelitis (especially in sickle cell disease where *Salmonella* is one of the most common pathogens)
- GI bleed/perforation
- meningitis
- cholecystitis
- chronic carriage (1%, more likely if adult females)



Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

6 9

Typhoid fever review

Suggest link Report broken link

Media



Salmonella - a quick introduction and overview

Let's Learn About Bugs - YouTube 4 0



Salmonella

ATP - YouTube 7 1



Salmonella

SketchyMedical - YouTube 6 3

Report broken media

Score: **16.8%**

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An 80-year-old lady presents from a nursing home with a one-day history of fevers, drowsiness and vomiting. She has been unwell, not eating for the past 3 days according to her carer. Recently she has been exhibiting behavioural changes, crying and agitation when lights are turned on in the room, and holding her neck. She has a back ground of Alzheimer's dementia, hypertension, congestive cardiac failure(CCF) and chronic kidney disease.

On examination, she is febrile with a temperature of 38.1oC. Heart rate 112bpm, respiratory rate 20breaths per minute and oxygen saturations of 94% on air. On auscultation, there are bibasal crepitations and cardiovascular examination is consistent with mild CCF. There is neck stiffness and photophobia on cranial nerve examination, pupils equal and reactive to light. Kernig's sign is positive.

Investigations:

Na+	129mmol/l
K+	4.8 mmol/l
Urea	10.9 mmol/l
Creatinine	123 µmol/l
Serum glucose	5.9mmol/l
C Reactive protein	95mg/l
Haemoglobin	126 g/l
White cell count	16.4 x 10^9/L
INR	1.2

Cerebro-spinal fluid (CSF) analysis:

Opening pressure	25 cmH20
Protein	1.8 g/L
Glucose	2.6 mmol/l
White cell count	>1000 per mm³
Gram Stain	Gram-positive rods seen
Colour	Cloudy, turbid

What is the likely causative organism?

- *Streptococcus pneumoniae*
- *Listeria monocytogenes*
- *Staphylococcus aureus*
- *Neisseria meningitidis*
- *Haemophilus influenzae*

Submit answer

Reference ranges

Score: **22%**

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Glucose	2.6 mmol/l
White cell count	>1000 per mm³
Gram Stain	Gram-positive rods seen
Colour	Cloudy, turbid

What is the likely causative organism?

Streptococcus pneumoniae	5%
Listeria monocytogenes	82%
Staphylococcus aureus	1%
Neisseria meningitidis	7%
Haemophilus influenzae	5%



This lady has bacterial meningitis as evidenced from her LP results. There is only one gram-positive bacilli in the list and that is *Listeria monocytogenes*, a common causative organism of meningitis in the extremes of age.

Gram-positive bacilli (or rods): ABCDL

- Actinomyces
- Bacillus anthracis
- Clostridium, Corynebacterium
- Diphtheria
- Listeria monocytogenes

Gram positive cocci:

- Streptococci
- Staphylococci

Gram-negative cocci: NNM

- Neisseria (Neisseria meningitides, Neisseria gonorrhoea)
- Moraxella

Gram negative bacilli (or rods): almost every other pathogenic gram negative bacteria.

- Salmonella spp.
- Escherichia spp.
- Pseudomonas
- Bacteroides

👍 👎 💬 Discuss (2) Improve

Next question ›

Listeria ★

Listeria monocytogenes is a Gram-positive bacillus which has the unusual ability to multiply at low temperatures. It is typically spread via contaminated food, typically unpasteurised dairy products. Infection is particularly dangerous to the unborn child where it can lead to miscarriage.

Risk factors

- elderly
- neonates
- immunosuppression
  - especially glucocorticoids
- pregnancy

Features - can present in a variety of ways

- gastroenteritis
  - diarrhoea
- bacteraemia
  - flu-like illness
- central nervous system infection
  - meningoencephalitis
  - ataxia
  - seizures

Investigation

- blood cultures
  - 'tumbling motility' on wet mounts
- cerebrospinal fluid findings:
  - pleocytosis, often lymphocytes (nontuberculous bacteria usually cause a rise in neutrophils)
  - raised protein
  - reduced glucose

Management

- Listeria is sensitive to amoxicillin/ampicillin (cephalosporins usually inadequate)
- Listeria meningitis should be treated with IV amoxicillin/ampicillin and gentamicin

In pregnant women

- pregnant women are almost 20 times more likely to develop listeriosis compared with the rest of the population due to changes in the immune system
- fetal/neonatal infection can occur both transplacentally and vertically during childbirth
- complications include miscarriage, premature labour, stillbirth and chorioamnionitis

- diagnosis can only be made from blood cultures
- treatment is with amoxicillin

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media



Tumbling motility of listeria

Individual - YouTube 22 3

Report broken media

Score: **16.8%**

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A 24-year-old male patient is referred to HIV clinic following positive testing on screening in a local GUM clinic. His CD4 count is 1020 cells/microlitre and viral load is 10,123 copies/ml. He is offered to start antiretroviral treatment or monitoring. He has a past medical history of depression and has emigrated from Nigeria three years ago. He is planning to return abroad in two years. What benefit has been established from starting early antiretroviral treatment as opposed to deferring treatment?

- Increased efficacy
- Reduced risk of AIDS and mortality
- Reduced drug resistance
- Reduced toxicity
- Reduced anxiety

Submit answer

Reference ranges

Score: **22%**

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Increased efficacy	6%
Reduced risk of AIDS and mortality	87%
Reduced drug resistance	6%
Reduced toxicity	1%
Reduced anxiety	1%

The correct answer is the reduced the risk of AIDS and mortality. BHIVA has made the recommendation to offer antiviral therapy regardless of the CD4 count. This is partly due to results of the SMART study which demonstrated reduced chance of AIDS, a serious non-AIDS event or death by 57% with similar results in high, middle and low-income countries. Data on virological efficacy, drug resistance and toxicity is not yet established. Reduced anxiety may also be a benefit but has not been as definitively established.

Source:
'BHIVA Guidelines for the Treatment of HIV-1-positive Adults with Antiretroviral Therapy 2015.' BHIVA. , Aug. 2016.

Discuss (1) | Improve

Next question

HIV: management

Antiretroviral therapy (ART) involves a combination of at least three drugs, typically two nucleoside reverse transcriptase inhibitors (NRTI) and either a protease inhibitor (PI) or a non-nucleoside

reverse transcriptase inhibitor (NNRTI). This combination both decreases viral replication but also reduces the risk of viral resistance emerging

Following the 2015 BHIVA guidelines it is now recommended that patients start ART as soon as they have been diagnosed with HIV, rather than waiting until a particular CD4 count, as was previously advocated.

Entry inhibitors

- maraviroc (binds to CCR5, preventing an interaction with gp41), enfuvirtide (binds to gp41, also known as a 'fusion inhibitor')
- prevent HIV-1 from entering and infecting immune cells

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), abacavir, emtricitabine, didanosine, lamivudine, stavudine, zalcitabine, tenofovir
- general NRTI side-effects: peripheral neuropathy
- tenofovir: used in BHIVAs two recommended regime NRTI. Adverse effects include renal impairment and ostesoporosis
- zidovudine: anaemia, myopathy, black nails
- didanosine: pancreatitis

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz
- side-effects: P450 enzyme interaction (nevirapine induces), rashes

Protease inhibitors (PI)

- examples: indinavir, nelfinavir, ritonavir, saquinavir
- side-effects: diabetes, hyperlipidaemia, buffalo hump, central obesity, P450 enzyme inhibition
- indinavir: renal stones, asymptomatic hyperbilirubinaemia
- ritonavir: a potent inhibitor of the P450 system

Integrase inhibitors

- block the action of integrase, a viral enzyme that inserts the viral genome into the DNA of the host cell
- examples: raltegravir, elvitegravir, dolutegravir

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

7 4

2016 HIV-1 infection guidelines

University of Liverpool

12 6

HIV Drug Interactions Checker

Suggest link

Report broken link

Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube

1 0



Antiretroviral drugs

Medinaz - YouTube

38 2

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Score: **16.8%**

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A 42-year-old man attends the GP with a lesion on his left hand. He says it first appeared as a red spot 2-3 weeks ago when he returned from Iraq. However, since then the lesion has been progressively enlarging. There is no history of pain or itch and he otherwise feels well. He is normally fit and well and takes no regular medications.

On examination, his observations are all within the normal range. His hand appears as shown below. On further inspection, there is sparing of the mucous membranes and no further lesions are found on his body.



What is the most likely diagnosis?

- Anthrax
- Chagas disease
- Leishmaniasis
- Leprosy
- Lyme disease

Submit answer

Reference ranges

Score: **22%**

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On examination, his observations are all within the normal range. His hand appears as shown below. On further inspection, there is sparing of the mucous membranes and no further lesions are found on his body.



What is the most likely diagnosis?

Anthrax	8%
Chagas disease	2%
Leishmaniasis	**84%**
Leprosy	5%
Lyme disease	0%

This patient has a superficial cutaneous lesion on his left hand with central ulceration. The surrounding skin is not affected. Given the history of rapid growth from a small red papule and a recent tour in Iraq, it is likely that this patient has leishmaniasis, a parasitic disease spread by

sandflies. There are different species of leishmania that are responsible for different manifestations of the disease, with cutaneous leishmaniasis being the most common. Initially, a small red papule appears at the site of the sandfly bite which gradually enlarges followed by central ulceration before spontaneously resolving. The time to resolution varies between 2 months and 1 year. The Middle East, North Africa, and Asia are more common areas for contracting the disease.

Anthrax is a bacterial disease caused by *Bacillus anthracis*. Infection is associated with exposure to infected carcasses and the introduction of the organism into the body through a superficial wound. An initial pustule appears at the site of infection which progresses to a black eschar (a necrotic-looking ulcer with a black scab). The infection usually resolves spontaneously, but in some can lead to death. The lesion shown in this question is not that of a black eschar but rather a crusted ulcer from a sandfly bite. This makes leishmaniasis the correct diagnosis.

Chagas disease is a parasitic disease spread by triatomine or 'kissing' bugs. The disease is prevalent in Latin America and consists of an initial acute phase and chronic phase. In the earlier stages of the disease, symptoms include generalised malaise, fever, headaches, and myalgia along with anorexia, nausea, and vomiting. In others, a chagoma (an indurated, oedematous skin lesion) may be seen. In contrast, the patient in this question has returned from the Middle East with a cutaneous ulcerated lesion, making Chagas disease unlikely.

Leprosy is a granulomatous disease caused by *Mycobacterium leprae* that affects the peripheral nerves and skin. Initial symptoms include numbness, typically with loss of temperature followed by light touch, pain, and finally deep pressure. Cutaneous lesions follow later in the disease and appear as hypopigmented patches of skin with associated sensory loss. This patient has a painless, crusted ulcer with no other lesions or neurology making leprosy an unlikely diagnosis.

Ticks are well-known to spread Lyme disease, a bacterial infection caused by *Borrelia burgdorferi* . The most common dermatological manifestation of Lyme disease is a bulls-eye rash that appears at the site of the tick bite. Other symptoms include fever, lethargy, and arthralgia that are not described in this question.

Discuss (1) | Improve

Next question >

Leishmaniasis

Leishmaniasis is caused by the intracellular protozoa *Leishmania*, which are spread by the bites of sandflies. Cutaneous, mucocutaneous leishmaniasis and visceral forms are seen

Cutaneous leishmaniasis

- caused by *Leishmania tropica* or *Leishmania mexicana*
- crusted lesion at the site of bite
- there may be an underlying ulcer

- it is typically diagnosed by doing a punch biopsy from the edge of the lesion allowing for both histology and culture
- cutaneous leishmaniasis acquired in South or Central America merits treatment due to the risk of mucocutaneous leishmaniasis whereas disease acquired in Africa or India can be managed more conservatively







Mucocutaneous leishmaniasis

- caused by *Leishmania braziliensis*
- skin lesions may spread to involve mucosae of nose, pharynx etc

Visceral leishmaniasis (kala-azar)

- mostly caused by *Leishmania donovani*
- occurs in the Mediterranean, Asia, South America, Africa
- fever, sweats, rigors
- massive splenomegaly. hepatomegaly
- poor appetite*, weight loss
  - occasionally patients may report increased appetite with paradoxical weight loss
- grey skin - 'kala-azar' means black sickness
- pancytopaenia secondary to hypersplenism
- the gold standard for diagnosis is bone marrow or splenic aspirate
- management
  - the BNF recommends sodium stibogluconate, an organic pentavalent antimony compound
  - amphotericin B may be used with or after treatment with an antimony compound

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 9 8

2011 Leishmaniasis review

DermNet NZ 9 6

Leishmaniasis review

Suggest link Report broken link

Media



What is Leishmaniasis? An introduction and overview

Let's Learn About Bugs - YouTube 5 2

Report broken media

Score: **16.8%**

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A 63 year old man, originally from Ukraine is admitted to hospital with a right sided pleural effusion. On admission he is febrile at 37.8 C. He reports 6kg of weight loss, and night sweats over the previous 4 months.

His pleural effusion is exudative with a pH of 7.4.

A CT chest/abdo/pelvis reveals a right sided pleural effusion, multiple enlarged lymph nodes in his abdomen and mediastinum. A pleural biopsy reveals scanty Acid Fast Bacilli and he is commenced on treatment for tuberculosis with Rifampicin, Isoniazid, Ethambutol, Pyrizinamide. PCR confirms this to be Mycobacterial Tuberculosis complex with wild type rpoB, katG, inhA genes. His HIV serology is positive and his CD4 count is 47 cells/mm3

One week following admission he is commenced on anti-retroviral treatment with Emtricitabine, Lamivudine and Efavirenz with Co-trimoxazole.

Three weeks later he becomes more unwell. He has daily fevers up to 39°C.

On examination he is febrile 38.1 with an associated tachycardia of 119 beats/min, his blood pressure is 112/67 mmHg and respiratory rate is 22 breaths/min. His oxygen saturations are 93%. He has reduced air entry at the right base, but no other abnormality on examinations.

His results are as following:

Hb	10.1 g/dl
MCV	82 fl
Platelets	97 * 10^9/l
WBC	7.8 * 10^9/l
Neutrophils	6.3 * 10^9/l
Lypmphocytes	1.2 * 10^9/l
Eosinophils	0.02 * 10^9/l
ESR	62 mm/hr

Bilirubin	9 µmol/l
ALP	312 u/l
ALT	153 u/l

γGT	456 u/l
Albumin	22 g/l
CRP	67 mg/l
Lactate	1.1 mmol/l

Other results were as follows;

Urea and electrolytes: normal.
Clotting screen: normal
Blood cultures: negative (x3)
Urinalysis: normal
ECHO: normal
CXR: right pleural effusion
USS abdo: several abdominal nodes visible, nil else.
Toxoplasmosis serology: negative
CMV: IgG negative IgM: negative
EBV: IgG positive IgM: negative
Cryptococcal antigen: Negative
Leishmania serology: negative

What is the most likely diagnosis?

- ○ Pneumococcal pneumonia ×
- ○ Drug resistant tuberculosis ×
- ○ Infectious mononucleosis ×
- ○ Immune reconstitution inflammatory syndrome ×
- ○ Burkitts Lymplhoma ×

Submit answer

Reference ranges

Score: **22%**

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EBV: IgG positive IgM: negative
Cryptococcal antigen: Negative
Leishmania serology: negative

What is the most likely diagnosis?

- Pneumococcal pneumonia 2%
- Drug resistant tuberculosis 4%
- Infectious mononucleosis 3%
- Immune reconstitution inflammatory syndrome 88%
- Burkitts Lymplhoma 3%

This is a classic history of Immune Reconstitution Inflammatory syndrome (IRIS). IRIS is a paradoxical worsening of symptoms, due to increasing inflammatory activity with a recovering immune system. It is seen in the context of anti-retroviral therapy.

In this case we are reassured of a correct diagnosis to TB with the PCR result, and the initial PCR results suggest a fully susceptible organism (although this will need to be confirmed by culture and sensitivity testing.) You may not have a luxury of a confident diagnosis of TB, and patients with HIV may have many different opportunistic infections. Therefore IRIS is a diagnosis of exclusion. In this case an extensive screen has revealed no alternative cause for his symptoms.

The diagnosis of IRIS depends on all three of :

1. Antecedants:

- Diagnosis of TB fulfilling WHO clinical criteria
- Initial response to anti-retroviral treatment

2. Clinical (1 major criteria or 2 minor)

Major: New or worsening cold abscess lymph nodes, radiological features of TB, CNS TB or TB serositis

Minor: New or worsening constitutional symptoms, respiratory symptoms or abdominal pain.

3. Alternative explanations for clinical deterioration must be excluded.

Discuss (6) Improve

Next question

Immune reconstitution inflammatory syndrome

Immune reconstitution inflammatory syndrome is a condition generally associated with HIV/immunosuppression, in which the immune system begins to recover, but then responds to a previously acquired opportunistic infection with an overwhelming inflammatory response that paradoxically makes the symptoms of infection worse.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British HIV Association (BHIVA) 2 0

Immune reconstitution inflammatory syndrome (IRIS) / paradoxical reactions

Suggest link Report broken link

Score: **16.8%**

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A 17 year old girl presents to A&E accompanied by her anxious mother. She was born in England, but both her parents were born in a rice growing community in rural China. She has recently returned from a holiday where she was visiting her family in China. They live on a farm where she had close contact with dogs, sheep and pigs.
She arrived home one week ago. She was taking Mefloquine for malaria prophylaxis.

She was complaining of fever and a headache yesterday. Today she has been confused and disorientated.

She is previously fit and well, she has had all her immunisations according to the UK immunisation schedule, with normal growth and development. She does well at college and is a keen member of her college canoe club. She has no allergies.

On examination she is febrile at 38.9°C but haemodynamically stable. She appears confused and has an obvious difficultly walking. Examination of her cranial nerves is difficult but unremarkable. She has normal power and increased tone in her arms, which is more pronounced on the left side, with hyperreflexia bilaterally. She has several writhing involuntary movements of her upper limbs during the consultation.

A CT scan shows hypodensity in the thalami and basal ganglia bilaterally, more pronounced on the left side.

A lumbar puncture reveals a lymphocytic CSF with a raised protein.

What is the most likely diagnosis?

- Herpes Simplex Encephalitis
- Japanese Encephalitis
- Alcohol consumption
- Rabies
- Mefloquine toxicity

Submit answer

Reference ranges

Score: **22%**

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A CT scan shows hypodensity in the thalami and basal ganglia bilaterally, more pronounced on the left side.

A lumbar puncture reveals a lymphocytic CSF with a raised protein.

What is the most likely diagnosis?

Herpes Simplex Encephalitis	11%
Japanese Encephalitis	83%
Alcohol consumption	1%
Rabies	5%
Mefloquine toxicity	2%

The most likely diagnosis in this case is **Japanese Encephalitis**. The patient's history of travel to a rural area in China, where she had close contact with animals, combined with her presenting symptoms of fever, headache, confusion, and difficulty walking, as well as the CT scan findings of hypodensity in the thalami and basal ganglia bilaterally are all consistent with Japanese Encephalitis. This disease is a mosquito-borne flavivirus infection that predominantly affects people living or travelling to Asia.

Herpes Simplex Encephalitis is less likely due to the patient's travel history and exposure to animals. Although Herpes Simplex Encephalitis can also present with fever, headache, and confusion, it typically has a more acute onset and would show different findings on imaging studies (such as temporal lobe involvement on CT or MRI).

Alcohol consumption is an unlikely cause for this patient's presentation. While excessive alcohol intake can lead to neurological symptoms such as ataxia and confusion, there is no indication from the patient's history that she has been consuming large amounts of alcohol. Furthermore, her CT scan findings and lumbar puncture results do not support this diagnosis.

Rabies could be considered given the patient's close contact with animals during her trip; however, her clinical presentation does not fit the classic signs of rabies. Rabies usually presents with hydrophobia (fear of water), hypersalivation, and progressive paralysis. Additionally, while rabies can affect the central nervous system leading to encephalitis-like symptoms, it would not produce the specific CT scan findings seen in this case.

Finally, **Mefloquine toxicity** is an unlikely diagnosis because although mefloquine can cause neuropsychiatric side effects like dizziness or insomnia in some individuals, the patient's symptoms and investigation results are much more consistent with Japanese Encephalitis. Mefloquine toxicity would not typically cause the neurological findings observed in this case, nor would it explain the CT scan and lumbar puncture results.

Discuss (4) Improve

Next question ›

Japanese encephalitis

Japanese encephalitis is the most common cause of viral encephalitis in South East Asia, China the Western Pacific and India, with approx. 50,000 cases annually. It is a flavivirus transmitted by culex mosquitos which breeds in rice paddy fields. The reservoir hosts are aquatic birds, but pigs are an amplification host and therefore close domestic contact with pigs is a risk factor.

The majority of infection is asymptomatic.

Clinical features are headache, fever, seizures and confusion. Parkinsonian features indicate basal ganglia involvement. It can also present with acute flaccid paralysis.

Diagnosis is by serology or PCR. Management is supportive.

Prevention is a vaccine and there are a variety of different types.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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Back to top

A 45-year-old male from Afghanistan presents with a rash all over his body. He describes the rash and suggests that it was initially multiple specks of light skin that were not raised or roughened. they have now progressed to plaques, some of which have become nodular.

He is otherwise well and his past medical history is significant only for the successful treatment of visceral leishmaniasis in his home country some 5 years ago.

What is the most likely underlying diagnosis?

- Leprosy
- HIV
- Post kala azar dermal leishmaniasis (PKDL)
- Histoplasmosis
- Pityriasis versicolor

Submit answer

Reference ranges

Score: **22%**

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Leprosy	26%
HIV	1%
Post kala azar dermal leishmaniasis (PKDL)	66%
Histoplasmosis	1%
Pityriasis versicolor	6%

AI

This is a classic description of post kala azar dermal leishmaniasis (PKDL) from an area where the disease is endemic. Leishmaniasis can manifest as cutaneous, muco-cutaneous and visceral disease. PKDL is a chronic skin condition that arises after the treatment of visceral disease. The skin condition can arise months or years after successful treatment and often presents with erythematous or hypo-pigmented macules that may progress to become nodular. Clinically the lesions look very similar to pityriasis versicolour, however this has a predilection for the chest and back and is likely to have a scaly appearance rather than nodular. Perhaps the most important differential is leprosy and lesions should be tested for anaesthesia which would be strongly indicative for leprosy over PKDL.

Although HIV can cause a myriad of skin pathology there is nothing given in the question to make this a likely diagnosis and the best answer is therefore PKDL.

Leishmaniasis

Leishmaniasis is caused by the intracellular protozoa *Leishmania*, which are spread by the bites of sandflies. Cutaneous, mucocutaneous leishmaniasis and visceral forms are seen

Cutaneous leishmaniasis

- caused by *Leishmania tropica* or *Leishmania mexicana*
- crusted lesion at the site of bite
- there may be an underlying ulcer
- it is typically diagnosed by doing a punch biopsy from the edge of the lesion allowing for both histology and culture
- cutaneous leishmaniasis acquired in South or Central America merits treatment due to the risk of mucocutaneous leishmaniasis whereas disease acquired in Africa or India can be managed more conservatively





Mucocutaneous leishmaniasis

- caused by *Leishmania braziliensis*
- skin lesions may spread to involve mucosae of nose, pharynx etc

Visceral leishmaniasis (kala-azar)

- mostly caused by *Leishmania donovani*
- occurs in the Mediterranean, Asia, South America, Africa
- fever, sweats, rigors
- massive splenomegaly. hepatomegaly
- poor appetite*, weight loss
    - occasionally patients may report increased appetite with paradoxical weight loss
- grey skin - 'kala-azar' means black sickness
- pancytopaenia secondary to hypersplenism
- the gold standard for diagnosis is bone marrow or splenic aspirate
- management
    - the BNF recommends sodium stibogluconate, an organic pentavalent antimony compound
    - amphotericin B may be used with or after treatment with an antimony compound

AI + Q 123

Next question

Textbooks

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Links

Royal College of Physicians 9 8

2011 Leishmaniasis review

DermNet NZ 9 6

Leishmaniasis review

Suggest link Report broken link

Media



What is Leishmaniasis? An introduction and overview

Let's Learn About Bugs - YouTube 5 2

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Score: **16.8%**

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A 40-year-old male presented to the Emergency Department with a decreasing level of consciousness. He had just returned from the Hajj pilgrimage in Saudi Arabia. He was completely healthy during his travels, but he started to develop a fever two days after his return. The fever was associated with a severe frontal headache and photophobia. These symptoms persisted for the last two days and he started to become sleepier.

On examination: blood pressure 100/80 mmHg, pulse rate 120/min, temperature 39.2°C, respiratory rate 27/min. He had evidence of neck stiffness. Blood investigations showed:

Hb	123 g/l
Platelets	130* 10^9/l
WBC	13* 10^9/l

What is the most likely diagnosis?

- Meningococcal meningits type A
- Meningococcal meningits type B
- Meningococcal meningits type C
- Malaria
- Hemorrhagic fever

Submit answer

Reference ranges

Score: **22%**

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What is the most likely diagnosis?

Meningococcal meningits type A	28%
Meningococcal meningits type B	29%
Meningococcal meningits type C	29%
Malaria	6%
Hemorrhagic fever	8%

The candidate should have no difficulty in recognising that this patient has meningitis. However, good knowledge of the epidemiology of meningitis is required to answer the question completely.

The most common cause of meningitis in Saudi Arabia during Hajj is meningococcal meningitis type A. It is also the leading cause of epidemics of what is known as the meningitis belt in Africa along the equator line including Sudan, Ethiopia and Nigeria.

Type B and Type C are mainly responsible for sporadic cases of meningitis in Europe and South America.

Source http://www.who.int/csr/resources/publications/meningitis/whoemcbac983.pdf

Meningitis: causes

0 - 3 months

- Group B *Streptococcus* (most common cause in neonates)
- *E. coli*
- *Listeria monocytogenes*

3 months - 6 years

- *Neisseria meningitidis*
- *Streptococcus pneumoniae*
- *Haemophilus influenzae*

6 years - 60 years

- *Neisseria meningitidis*
- *Streptococcus pneumoniae*

> 60 years

- *Streptococcus pneumoniae*
- *Neisseria meningitidis*
- *Listeria monocytogenes*

Immunosuppressed

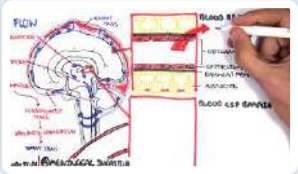
- *Listeria monocytogenes*

Textbooks

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Media



(Bacterial) Meningitis Pathophysiology

Armando Hasudungan - YouTube 2 0



Meningitis

Osmosis - YouTube 2 0

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A 54-year-old intravenous drug user is newly diagnosed with HIV, after presenting with progressive shortness of breath. He was diagnosed with pneumocystis pneumonia and commenced on appropriate treatment.

His CD4 count is 54 cells/mm3 and his viral load is 1.7 x10^7 copies per ml.

As part of his routine workup, it is also revealed that he has co-infection with hepatitis C. A test for HIV viral tropism is reported as dual tropism virus.

Which drug is unlikely to be effective in this man's treatment?

- Maraviroc
- Raltegravir
- Tenofovir
- Ribavirin
- Abacavir

Submit answer

Reference ranges

Score: **22%**

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As part of his routine workup, it is also revealed that he has co-infection with hepatitis C. A test for HIV viral tropism is reported as dual tropism virus.

Which drug is unlikely to be effective in this man's treatment?

Maraviroc	28%
Raltegravir	8%
Tenofovir	25%
Ribavirin	28%
Abacavir	11%

AI

HIV binds to the CD4 cell via the CD4 receptor. This interaction also depends on viral interaction with the CD4 co-receptor. There are two forms of CD4 co-receptor CCR5 and CXCR4.

The test for viral tropism determines which of these co-receptors HIV will bind to. Maraviroc blocks HIV binding to the CCR5 receptor, and therefore is an effective drug in a 'CCR5 tropic' virus. However, it is not effective if the virus is 'CXCR4 tropic' or 'dual tropic'. The tropism of HIV will not affect the use of any of the other drugs listed.

It is routine to test HIV viral tropism on diagnosis, or on virological failure to assess if maraviroc is a therapeutic option.

HIV: management

Antiretroviral therapy (ART) involves a combination of at least three drugs, typically two nucleoside reverse transcriptase inhibitors (NRTI) and either a protease inhibitor (PI) or a non-nucleoside reverse transcriptase inhibitor (NNRTI). This combination both decreases viral replication but also reduces the risk of viral resistance emerging

Following the 2015 BHIVA guidelines it is now recommended that patients start ART as soon as they have been diagnosed with HIV, rather than waiting until a particular CD4 count, as was previously advocated.

Entry inhibitors

- maraviroc (binds to CCR5, preventing an interaction with gp41), enfuvirtide (binds to gp41, also known as a 'fusion inhibitor')
- prevent HIV-1 from entering and infecting immune cells

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), abacavir, emtricitabine, didanosine, lamivudine, stavudine, zalcitabine, tenofovir
- general NRTI side-effects: peripheral neuropathy
- tenofovir: used in BHIVAs two recommended regime NRTI. Adverse effects include renal impairment and ostesoporosis
- zidovudine: anaemia, myopathy, black nails
- didanosine: pancreatitis

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz
- side-effects: P450 enzyme interaction (nevirapine induces), rashes

Protease inhibitors (PI)

- examples: indinavir, nelfinavir, ritonavir, saquinavir
- side-effects: diabetes, hyperlipidaemia, buffalo hump, central obesity, P450 enzyme inhibition
- indinavir: renal stones, asymptomatic hyperbilirubinaemia
- ritonavir: a potent inhibitor of the P450 system

Integrase inhibitors

- block the action of integrase, a viral enzyme that inserts the viral genome into the DNA of the host cell
- examples: raltegravir, elvitegravir, dolutegravir

Next question

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Links

BHIVA

2016 HIV-1 infection guidelines

7 4

University of Liverpool

HIV Drug Interactions Checker

12 6

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Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube

1 0



Antiretroviral drugs

Medinaz - YouTube

38 2

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On examination, there is a handful of atrophic scars over his cheeks, arms, and ankles. There are also raised scaly lesions with interspersed erythematous ulcerated papules, some of which are oozing pus in the same distribution.

What diagnostic tool would typically be used to confirm the likely diagnosis?

Blood cultures	3%
Bone marrow aspiration	6%
Punch biopsy of a lesion	60%
Skin scraping of a lesion	23%
Splenic aspirate	9%

Cutaneous leishmaniasis is typically diagnosed using a punch biopsy

Important for me Less important

The most likely diagnosis here is cutaneous leishmaniasis. This should be suspected in any patient with non-healing, scaling skin lesions, particularly if they have been in an endemic area.

Punch biopsy is correct. This is the typical method of diagnosing cutaneous leishmaniasis.

Blood cultures is incorrect. Leishmaniasis is a parasitic disease, blood culture is therefore not the typical diagnostic tool. In some cases, evidence of leishmaniasis can be seen on blood film.

Bone marrow aspiration is incorrect. This would be a diagnostic tool if we were concerned about visceral leishmaniasis. Fevers, substantial hepatomegaly, and blackening of the skin would raise suspicion of visceral leishmaniasis. None of these features are mentioned in the stem for this patient.

Skin scraping of a lesion is incorrect. Generally, leishmaniasis is diagnosed with PCR testing of a punch biopsy. This can also be performed on a sample obtained through skin scraping, but the diagnostic sensitivity is much poorer.

Splenic aspirate is incorrect. This would be a diagnostic tool if we were concerned about visceral leishmaniasis. Fevers, substantial hepatomegaly, and blackening of the skin would raise suspicion of visceral leishmaniasis. None of these features are mentioned in the stem for this patient.

Discuss (1) | Improve

Next question >

Leishmaniasis

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Media



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Let's Learn About Bugs - YouTube 5 2

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Score: **16.8%**

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- Bone marrow aspiration
- Punch biopsy of a lesion
- Skin scraping of a lesion
- Splenic aspirate

Submit answer

Reference ranges

Score: **22%**

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A 60-year-old man presented with history of excessive urination especially during night which disturbs his sleep, excessive thirst and tiredness for the past 2 months. He was a known HIV positive patient for which he was on highly active anti-retroviral therapy (HAART) since 6 months. Clinical examination was within normal limits. HIV related diabetes mellitus was suspected by the treating physician. His blood investigations are as follows-

Hb	150 g/l
Platelets	150 * 10^9/l
WBC	7 * 10^9/l
Fasting blood glucose	10 mmol/l
Post prandial blood glucose	13.9 mmol/l
Serum creatinine	130 umol/L

He was planned to be started on metformin therapy. Which of the following investigations is important to be checked before starting metformin in this patient?.

Liver enzymes	17%
Venous lactate	75%
Serum triglycerides	4%
Serum albumin	1%
Serum ammonia	2%

Patients with HIV are at increased risk of lactic acidosis and hence it is prudent to check lactate levels before starting metformin in such patients.Lactic acidaemia with no or mild symptoms was detected in 8% to 21% of patients receiving at least 1 nucleoside reverse transcriptase inhibitor, versus 0% to 1% of patients receiving no antiretroviral therapy. Symptomatic lactic acidaemia is less common (occurring in about 1.5%2.5%).Monitoring for lactic acidosis is recommended in the initial few months of metformin therapy. Patients with impaired renal function(serum creatinine>1.5 mg/dl) or with a venous lactate level more than twice the normal level prior to treatment should not be started on metformin.

This patient has an elevated serum creatinine but not >1.5mg/dl. So, he should get a venous lactate estimation before being started on metformin therapy considering the evidences so far.

Reference:
1) Clinical medicine 2014 Vol 14, No 6: 667-9
2) http://www.intmedpress.com/serveFile.cfm?sUID=f0b9cf79-9ed3-41da-b1b5-57a2bbae687a
3)http://journals.lww.com/jaids/Abstract/2002/11010/ManagementofMetabolicComplicationsAssociated.1.as
(Management of Metabolic Complications Associated With Antiretroviral Therapy for HIV-1
Infection: Recommendations of an International AIDS SocietyUSA Panel)

Discuss (6) | Improve

Next question >

HIV: management ★

Antiretroviral therapy (ART) involves a combination of at least three drugs, typically two nucleoside reverse transcriptase inhibitors (NRTI) and either a protease inhibitor (PI) or a non-nucleoside reverse transcriptase inhibitor (NNRTI). This combination both decreases viral replication but also reduces the risk of viral resistance emerging

Following the 2015 BHIVA guidelines it is now recommended that patients start ART as soon as they have been diagnosed with HIV, rather than waiting until a particular CD4 count, as was previously advocated.

Entry inhibitors

- maraviroc (binds to CCR5, preventing an interaction with gp41), enfuvirtide (binds to gp41, also known as a 'fusion inhibitor')
- prevent HIV-1 from entering and infecting immune cells

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), abacavir, emtricitabine, didanosine, lamivudine, stavudine, zalcitabine, tenofovir
- general NRTI side-effects: peripheral neuropathy
- tenofovir: used in BHIVAs two recommended regime NRTI. Adverse effects include renal impairment and ostesoporosis
- zidovudine: anaemia, myopathy, black nails
- didanosine: pancreatitis

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz
- side-effects: P450 enzyme interaction (nevirapine induces), rashes

Protease inhibitors (PI)

- examples: indinavir, nelfinavir, ritonavir, saquinavir
- side-effects: diabetes, hyperlipidaemia, buffalo hump, central obesity, P450 enzyme inhibition
- indinavir: renal stones, asymptomatic hyperbilirubinaemia

- ritonavir: a potent inhibitor of the P450 system

Integrase inhibitors

- block the action of integrase, a viral enzyme that inserts the viral genome into the DNA of the host cell
- examples: raltegravir, elvitegravir, dolutegravir

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

2016 HIV-1 infection guidelines

7 4

University of Liverpool

HIV Drug Interactions Checker

12 6

Suggest link Report broken link

Media



Treating HIV: Antiretroviral drugs

Khan Academy - YouTube

4 0



HIV antiretroviral drugs (classes, mechanism of action and side effects)

Armando Hasudungan - YouTube

1 0



Antiretroviral drugs

Medinaz - YouTube

38 2

Report broken media

Score: **16.8%**

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A 60-year-old man presented with history of excessive urination especially during night which disturbs his sleep, excessive thirst and tiredness for the past 2 months. He was a known HIV positive patient for which he was on highly active anti-retroviral therapy (HAART) since 6 months. Clinical examination was within normal limits. HIV related diabetes mellitus was suspected by the treating physician. His blood investigations are as follows-

Hb	150 g/l
Platelets	150 * 10^9/l
WBC	7 * 10^9/l
Fasting blood glucose	10 mmol/l
Post prandial blood glucose	13.9 mmol/l
Serum creatinine	130 umol/L

He was planned to be started on metformin therapy. Which of the following investigations is important to be checked before starting metformin in this patient?.

- Liver enzymes
- Venous lactate
- Serum triglycerides
- Serum albumin
- Serum ammonia

Submit answer

Reference ranges

Score: **22%**

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A 25-year-old man reports feeling unwell with fever, generalised muscle aches, headache and widespread rash 1 week after returning from Thailand. On admission his chest X-ray is unremarkable. His urine dip showed 1+ protein only and viral swabs are negative. Bloods are also normal aside from low platelet count. On day 2 of admission his blood pressure drops and repeat bloods show a further fall in platelets and reduction in haemoglobin count. What is the most likely diagnosis?

Measles	1%
Malaria	8%
Dengue fever	86%
Gonococcal septicaemia	4%
Herpes simplex encephalitis	1%

Retro-orbital headache, fever, facial flushing, rash, thrombocytopenia in returning traveller → ?dengue

Important for me Less important

All plausible options and further history including prior vaccination, sexual history and malaria prophylaxis would be helpful. The history and deterioration are most likely due to dengue fever and subsequent 'dengue shock syndrome'. Measles would be unlikely at his age, malaria does not classically cause a widespread rash and herpes simplex would not cause a drop in the platelets. Gonorrhoea fits quite well and has a similar incubation period (4-7 days) . The patient would have normally have reported an isolated septic joint by this stage in the disease.

Discuss (2) Improve

Next question

Dengue fever

Dengue fever is a viral infection that can progress to viral haemorrhagic fever (other examples include yellow fever, Lassa fever, Ebola).

Aetiology

- dengue virus is a RNA virus of the genus Flavivirus
- transmitted by the *Aedes aegypti* mosquito
- incubation period of 7 days

Clinical features

Patients with dengue fever can be classified as follows:

- dengue fever:
  - without warning signs
  - with warning signs
- severe dengue (dengue haemorrhagic fever)

Dengue fever

- fever
- headache (often retro-orbital)
- myalgia, bone pain and arthralgia ('break-bone fever')
- pleuritic pain
- facial flushing (dengue)
- maculopapular rash
- haemorrhagic manifestations e.g. positive tourniquet test, petechiae, purpura/ecchymosis, epistaxis
- 'warning signs' include:
  - abdominal pain
  - hepatomegaly
  - persistent vomiting
  - clinical fluid accumulation (ascites, pleural effusion)

Severe dengue (dengue haemorrhagic fever)

- this is a form of disseminated intravascular coagulation (DIC) resulting in:
  - thrombocytopenia
  - spontaneous bleeding
- around 20-30% of these patients go on to develop dengue shock syndrome (DSS)

Investigations

- typically blood results
  - leukopenia, thrombocytopenia, raised aminotransferases
- diagnostic tests
  - serology
  - nucleic acid amplification tests for viral RNA

- NS1 antigen test

Treatment

- entirely symptomatic e.g. fluid resuscitation, blood transfusion etc
- no antivirals are currently available

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 8 14

2011 Dengue fever review

Postgraduate Medical Journal 6 9

Review of dengue fever

Suggest link Report broken link

Score: **16.8%**

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- Measles
- Malaria
- Dengue fever
- Gonococcal septicaemia
- Herpes simplex encephalitis

Submit answer

Reference ranges

Score: **22%**

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A 68-year-old lady presents with lower abdominal pain, dysuria, and urinary frequency. A urine dip was positive for nitrites and leukocytes. She has a past medical history of recurrent C.difficile infection which has been treated with two tapering courses of vancomycin.

Microbiology results are as follows:

Urine culture	Vancomycin Resistant Enterococcus (VRE)

How will you manage this patient?

Amoxicillin	3%
Linezolid	85%
Gentamicin	10%
Penicillin	1%
Erythromycin	1%

Vancomycin Resistant Enterococcus (VRE) can be treated with linezolid, daptomycin and tigecycline

Important for me Less important

Enterococci are gram positive gamma-haemolytic Streptococci. Most important are E. faecalis and E. faecium. They are part of the normal flora of the human intestine, the female genital tract, and urinary tract. They are intrinsically of low pathogenicity however have a natural high resistance to antibiotics.

Vancomycin Resistant Enterococcus (VRE) are important nosocomial pathogens for which there are limited treatment options. Predisposing factors for VREs include previous treatment with anti-microbials (especially vancomycin, cephalosporins and broad-spectrum antibiotics).

Most VRE have high-level resistance to β-lactams and aminoglycosides.

Management of VRE may include removal of catheters and drainage of abscesses

If after clinical assessment and primary management, antimicrobial treatment of VRE is considered necessary, a drug should be selected based upon susceptibility testing of the organism involved. Nearly all strains of E. faecalis, including VRE strains, are susceptible to ampicillin. VREs also often remain sensitive to chloramphenicol, rifampicin and tetracyclines. A number of newer agents include linezolid, daptomycin and tigecycline.

Gentamicin can be used synergistically with another antibiotic, however it should not be used alone.

Discuss (5) Improve

Next question

Vancomycin

Vancomycin is a glycopeptide antibiotic used in the treatment of Gram-positive infections, particularly methicillin-resistant *Staphylococcus aureus* (MRSA).

Mechanism of action

- inhibits cell wall formation by binding to D-Ala-D-Ala moieties, preventing polymerization of peptidoglycans

Mechanism of resistance

- alteration to the terminal amino acid residues of the NAM/NAG-peptide subunits (normally D-alanyl-D-alanine) to which the antibiotic binds

Adverse effects

- nephrotoxicity
- ototoxicity
- thrombophlebitis
- red man syndrome; occurs on rapid infusion of vancomycin

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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A 26-year-old woman presents to the Emergency Department with a one week history of cough, fever and headache. She returned from Indonesia one week ago.

On examination she appears drowsy and a little muddled. She has no photophobia or focal neurological deficit. She has mild hepatosplenomegaly but no palpable lymphadenopathy. Her chest is clear and heart sounds are normal. She has a macular rash on both legs with a greyish-black scab-like lesion on her left shin.

Investigations results:

Chest x-ray: Bilateral patchy consolidation

Blood culture: Pending

Hb	120 g/l
Platelets	130 * 10^9/l
WBC	18 * 10^9/l
Blood film	Left shifted neutrophils in large numbers

Na^+	142 mmol/l
K^+	4.1 mmol/l
Urea	6 mmol/l
Creatinine	90 µmol/l

Bilirubin	23 µmol/l
ALP	170 u/l
ALT	50 u/l
Albumin	35 g/l

What is the most appropriate first line treatment?

- Azithromycin
- Chloroquine
- Doxycycline

- Mefloquine
- Supportive medications

Submit answer

Reference ranges

Score: **22%**

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Creatinine	90 µmol/l

Bilirubin	23 µmol/l
ALP	170 u/l
ALT	50 u/l
Albumin	35 g/l

What is the most appropriate first line treatment?

Azithromycin	9%
Chloroquine	3%
Doxycycline	79%

Mefloquine	4%
Supportive medications	5%

Doxycycline is typically used in the management of typhus

Important for me Less important

This lady has a triad of fever, hepatosplenomegaly and encephalitis, which in a returning traveller could point towards a number of diseases including malaria and dengue fever. However, the lesion on her shin is an eschar, which points towards a diagnosis of rickettsia. Given her clinical signs and that she has been to Indonesia, this is likely to be scrub typhus (infection with Orienta Tsutsugamushi). Low platelets, raised LFTs and bilateral infiltrates on chest x-ray support this diagnosis.

First line treatment is with doxycycline for seven days, although a single dose of azithromycin may work in those for whom it is not possible to give doxycycline.

Mefloquine and Chloroquine are used in malaria. Supportive therapy is the recommended treatment in dengue fever.

Discuss (4) Improve

Next question

Typhus

Typhus is a group of rickettsial diseases transmitted between hosts by arthropods. Examples include

- endemic typhus
  - caused by *Rickettsia typhi*
  - reservoir/vector: fleas on rats
  - occurs throughout the world, typically in warm costal regions
  - relative bradycardia
- epidemic typhus
  - caused by *Rickettsia prowazekii*
  - reservoir/vector: body louse
  - more common in central and eastern Africa, central and South America
- scrub typhus
  - caused by *Orientia tsutsugamushi*
  - reservoir/vector: harvest mites on humans or rodents

    - more common in Asia
    - black eschar at site of original inoculation
    - relative bradycardia despite fever
- spotted fever
    - caused by Rickettsia spotted fever group
    - spread by ticks
    - examples include Rocky Mountain spotted fever

Common features

- fever, headache, malaise
- rash
    - typically maculopapular
    - begins on the trunk and spreads to the extremities
- later complications
    - meningoencephalitis

Management

- doxycycline



Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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You are asked to review a patient on the ward who the nurse feels is looking very flushed. The nurse noticed the change in the patient whilst they were receiving their first dose of vancomycin. On examination the patient has no symptoms or signs of cardiorespiratory distress. Observations are as follows:

Temperature 37.2 ºc
Respiratory rate 18 breaths/min
Saturations on air 97%
Heart rate 70 beats/min
Blood Pressure 136/72 mmHg

A blanching macular rash is evident on the patient's upper arms and upper thighs. No signs of urticaria or excoriations. The patient is penicillin allergic.

What is your next step?

- Measure mast cell tryptase levels
- Stop the infusion, give the patient piriton and hydrocortisone
- Stop the infusion and give the patient piriton, hydrocortisone, adrenaline
- Continue the infusion but slow it down and give the patient piriton
- Stop the infusion, do not give any further treatment but discuss an alternative antibiotic with microbiology

Submit answer

Reference ranges

Score: **22%**

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You are asked to review a patient on the ward who the nurse feels is looking very flushed. The nurse noticed the change in the patient whilst they were receiving their first dose of vancomycin. On examination the patient has no symptoms or signs of cardiorespiratory distress. Observations are as follows:

Temperature 37.2 ºc
Respiratory rate 18 breaths/min
Saturations on air 97%
Heart rate 70 beats/min
Blood Pressure 136/72 mmHg

A blanching macular rash is evident on the patient's upper arms and upper thighs. No signs of urticaria or excoriations. The patient is penicillin allergic.

What is your next step?

Measure mast cell tryptase levels	2%
Stop the infusion, give the patient piriton and hydrocortisone	13%
Stop the infusion and give the patient piriton, hydrocortisone, adrenaline	1%
Continue the infusion but slow it down and give the patient piriton	69%
Stop the infusion, do not give any further treatment but discuss an alternative antibiotic with microbiology	14%

The most likely explanation for the patient's condition is a drug reaction called red man syndrome secondary to vancomycin infusion. This is not an allergic reaction as demonstrated by the lack of cardiorespiratory distress, subcutaneous oedema, wheals or urticaria. The reaction is more of a rate dependent infusion reaction. Treatment involves slowing down the infusion of vancomycin and administration of anti-histamines.

Further information about red many syndrome can be found at the following sources:

BNF: https:www.evidence.nhs.uk/formulary/bnf/current/5-infections/51-antibacterial-drugs/517-some-other-

antibacterials/glycopeptides/vancomycinhttp:www.uptodate.com/contents/vancomycin-hypersensitivity

Discuss (5) Improve

Next question ›

Vancomycin

Vancomycin is a glycopeptide antibiotic used in the treatment of Gram-positive infections, particularly methicillin-resistant *Staphylococcus aureus* (MRSA).

Mechanism of action

- inhibits cell wall formation by binding to D-Ala-D-Ala moieties, preventing polymerization of peptidoglycans

Mechanism of resistance

- alteration to the terminal amino acid residues of the NAM/NAG-peptide subunits (normally D-alanyl-D-alanine) to which the antibiotic binds

Adverse effects

- nephrotoxicity
- ototoxicity
- thrombophlebitis
- red man syndrome; occurs on rapid infusion of vancomycin

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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A 75-year-old male with a long history of intravenous drug use is admitted with fevers, rigors and back pain. Three sets of blood cultures taken at admission grow positive for gram positive cocci in clusters. He is suspected of having *Staphylococcus aureus* bacteraemia and is commenced on intravenous vancomycin.

Half an hour after the infusion is commenced, he is noted by the nurse to be flushed. On examination, he is noted to have erythema over his neck, face and trunk but denies any significant distress or discomfort.

His observations are as follow: blood pressure 125/70 mmHg, heart rate 85/min, temperature of 36.8ºC, respiratory rate of 18/min and oxygen saturation of 98% on room air.

Which of the following is the most appropriate management?

- Stopping the vancomycin infusion, administering 200mg of IV hydrocortisone and informing the patient that he is allergic to the medication
- Stopping the vancomycin infusion and administering a single dose of 0.5mcg intramuscular adrenaline
- Stopping the vancomycin infusion until symptoms resolve and then re-starting a slower rate
- Stopping the vancomycin infusion and prescribing topical 1% hydrocortisone cream to affected areas
- Continuing the vancomycin infusion and administering 1000 ml of 0.9% saline solution over 1 hour

Submit answer

Reference ranges

Score: **22%**

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His observations are as follow: blood pressure 125/70 mmHg, heart rate 85/min, temperature of 36.8ºC, respiratory rate of 18/min and oxygen saturation of 98% on room air.

Which of the following is the most appropriate management?

Stopping the vancomycin infusion, administering 200mg of IV hydrocortisone and informing the patient that he is allergic to the medication	6%
Stopping the vancomycin infusion and administering a single dose of 0.5mcg intramuscular adrenaline	2%
Stopping the vancomycin infusion until symptoms resolve and then re-starting a slower rate	75%
Stopping the vancomycin infusion and prescribing topical 1% hydrocortisone cream to affected areas	1%
Continuing the vancomycin infusion and administering 1000 ml of 0.9% saline solution over 1 hour	16%

Red man syndrome is associated with rapid intravenous infusion vancomycin. It is a common adverse reaction of intravenous vancomycin use and is a distinct entity from anaphylaxis due to vancomycin use. Typical symptoms include redness, pruritus and a burning sensation, predominantly in the upper body (face, neck and upper chest). Severe cases can be associated with hypotension and chest pain.

The pathophysiology of red man syndrome is attributed to vancomycin-related activation of mast cells with release of histamine.

The management of red man syndrome involves cessation of the infusion, and when symptoms have resolved, recommencement at a slower rate. In patients who are more symptomatic antihistamines can be administered, and may require intravenous fluids if the syndrome is associated with hypotension.

References:

- Sivagnanam S, Deleu D, Red man syndrome. Crit Care. 2003 Apr;7(2):119-20
- Bruneira FR, Ferreira FM, Saviolli LR, Bacci MR, Feder D, et al. The use of vancomycin with its therapeutic and adverse effects: a review Eur Rev Med Pharmacol Sci. 2015 Feb;19(4):694-700.

Discuss (4) Improve

Next question

Vancomycin

Vancomycin is a glycopeptide antibiotic used in the treatment of Gram-positive infections, particularly methicillin-resistant *Staphylococcus aureus* (MRSA).

Mechanism of action

- inhibits cell wall formation by binding to D-Ala-D-Ala moieties, preventing polymerization of peptidoglycans

Mechanism of resistance

- alteration to the terminal amino acid residues of the NAM/NAG-peptide subunits (normally D-alanyl-D-alanine) to which the antibiotic binds

Adverse effects

- nephrotoxicity
- ototoxicity
- thrombophlebitis
- red man syndrome; occurs on rapid infusion of vancomycin

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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You are the medical basic specialist trainee on call and have been asked to see a gentleman who has just been admitted for a bad chest infection. According to the ward nurse, he has developed an allergic reaction after five minutes of vancomycin i.v infusion.

On arrival you proceed to examine this young man who is febrile, obviously unwell and receiving oxygen via nasal prongs. He claims he had not developed any new symptoms since admission but felt sudden warmth and flushing few minutes into his vancomycin drip. On examination he has crepitations in his left base.

Apart from generalised erythema he appears to be stable and there is no immediate danger to his airways. His blood pressure was 105/67mmHg, pulse was 99b/min and oxygen saturation on 4L oxygen was 95% (all observations are similar to previous readings). He has never taken vancomycin but states he is allergic to penicillin and ciprofloxacin.

Given the likely diagnosis, what is the next step in your management?

- Immediately stop vancomycin
- Continue vancomycin but at a slower rate
- Continue vancomycin rate unchanged
- Administer i.v chlorphenamine maleate and stop vancomycin
- Urgent i.m adrenaline

Submit answer

Reference ranges

Score: **22%**

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Given the likely diagnosis, what is the next step in your management?

Immediately stop vancomycin	8%
Continue vancomycin but at a slower rate	77%
Continue vancomycin rate unchanged	2%
Administer i.v chlorphenamine maleate and stop vancomycin	10%
Urgent i.m adrenaline	2%

Answer: continue vancomycin but at a slower rate.

This is typical red man syndrome caused by rapid infusion of vancomycin i.v. Vancomycin should be administered diluted and at a rate of 10mg/minutes for doses over 500mg i.v.

It is an IgE independent mast cell degranulation reaction that occurs up to 10 minutes into receiving vancomycin i.v at a fast rate. It is not a true allergic reaction thus vancomycin should be continued but at a lower rate of infusion.

Antihistamine prophylaxis, lower but frequent dosing and 2 hour infusion rate have been found to reduce the incidence of red man syndrome.

This gentleman is obviously unwell but his vitals and chest findings are more in keeping with his chest infection and possible sepsis.

Red-man syndrome after vancomycin: potential cross-reactivity with teicoplanin C Khurana, M A de Belder http://pmj.bmj.com/content/75/879/41.long

Discuss (11) | Improve

Next question ›

Vancomycin

Vancomycin is a glycopeptide antibiotic used in the treatment of Gram-positive infections, particularly methicillin-resistant *Staphylococcus aureus* (MRSA).

Mechanism of action

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Mechanism of resistance

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Adverse effects

- nephrotoxicity
- ototoxicity
- thrombophlebitis
- red man syndrome; occurs on rapid infusion of vancomycin

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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A 36-year-old man is admitted with a painful right arm. On examination, there is mild erythema overlying the forearm but extreme tenderness to palpate. The arm is also tender beyond the area of erythema. Observations are as follows.

- Respiratory rate 32/min
- Oxygen saturation 94% on air
- Heart rate 134/min
- Blood pressure 72/44mmHg
- Temperature 38.4ºC

Over the next few hours, he deteriorates rapidly despite antibiotic therapy. The arm develops bullae and necrotic areas and ultimately requires amputation. His family arrive and ask how he could have developed this illness.

What recent infection could increase the risk of his current condition?

- Chickenpox
- Herpes simplex virus encephalitis
- Influenza
- Mumps
- SARS-CoV2

Submit answer

Reference ranges

Score: **22%**

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Over the next few hours, he deteriorates rapidly despite antibiotic therapy. The arm develops bullae and necrotic areas and ultimately requires amputation. His family arrive and ask how he could have developed this illness.

What recent infection could increase the risk of his current condition?

Chickenpox	75%
Herpes simplex virus encephalitis	6%
Influenza	5%
Mumps	6%
SARS-CoV2	8%

Chickenpox is a risk factor for invasive group A streptococcal soft tissue infections including necrotizing fasciitis

Important for me Less important

The correct answer is **chickenpox**. This patient has developed necrotising soft tissue infection, most commonly due to infection with group A streptococcus. Recent chickenpox is a recognised risk factor for invasive group A streptococcal (iGAS) infections such as this.

Herpes simplex virus encephalitis is incorrect. This is a severe central nervous system infection which does have complications including stroke and epilepsy. It is not known to increase the risk of iGAS.

Influenza is incorrect. The main bacterial infection seen in the aftermath of influenza is *Staphylococcus aureus* pneumonia.

Mumps is incorrect. It does have a range of complications including encephalitis and pancreatitis, but an increased risk of iGAS is not recognised.

SARS-CoV2 is incorrect. Infection with this virus can increase the risk of secondary bacterial respiratory tract infections. Some treatments also increase the risk of infections - for example, some areas saw increased rates of invasive fungal infections like mucormycosis related to high-dose corticosteroid use. An association with iGAS, however, has not been described.

Discuss (1) | Improve

Next question >

Chickenpox ★

Chickenpox is caused by primary infection with varicella zoster virus. Shingles is a reactivation of the dormant virus in dorsal root ganglion

Chickenpox is highly infectious

- spread via the respiratory route
- can be caught from someone with shingles
- infectivity = 4 days before rash, until 5 days after the rash first appeared*
- incubation period = 10-21 days

Clinical features (tend to be more severe in older children/adults)

- fever initially
- itchy, rash starting on head/trunk before spreading. Initially macular then papular then vesicular
- systemic upset is usually mild

Management is supportive

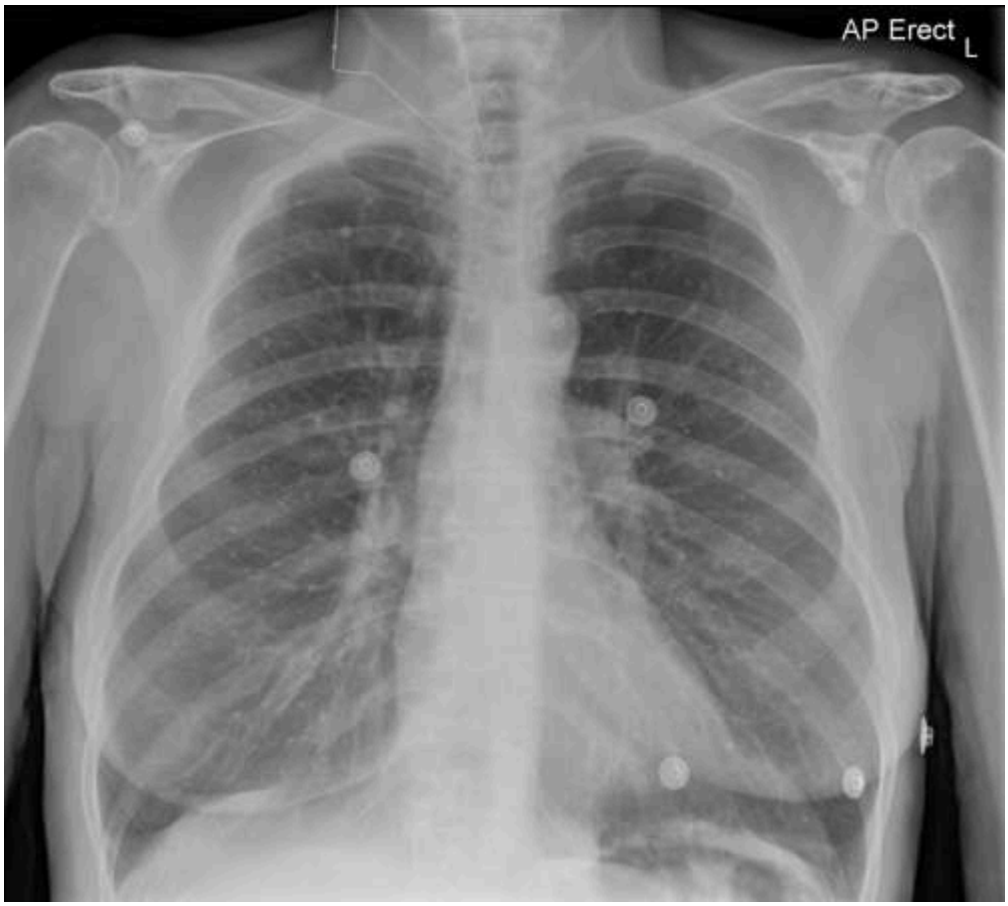
- keep cool, trim nails
- calamine lotion
- school exclusion: NICE state the following: *Advise that the most infectious period is 1-2 days before the rash appears, but infectivity continues until all the lesions are dry and have crusted over (usually about 5 days after the onset of the rash).*
- immunocompromised patients and newborns with peripartum exposure should receive varicella zoster immunoglobulin (VZIG). If chickenpox develops then IV aciclovir should be considered

A common complication is secondary bacterial infection of the lesions

- NSAIDs may increase this risk
- whilst this commonly may manifest as a single infected lesion/small area of cellulitis, in a small number of patients invasive group A streptococcal soft tissue infections may occur resulting in necrotizing fasciitis

Rare complications include

- pneumonia
- encephalitis (cerebellar involvement may be seen)
- disseminated haemorrhagic chickenpox
- arthritis, nephritis and pancreatitis may very rarely be seen



© Image used on license from Radiopaedia

Chest x-ray showing miliary opacities secondary to healed varicella pneumonia. Multiple tiny calcific miliary opacities noted throughout both lungs. These are of uniform size and dense suggesting calcification. There is no focal lung parenchymal mass or cavitating lesion seen. The appearances are characteristic for healed varicella pneumonia.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Greenbook 8 6

Varicella

Public Health England 9 6

Chapter 9: managing specific infectious diseases

Suggest link Report broken link

Media



Varicella zoster virus

Osmosis - YouTube 6 0

Report broken media

Score: **16.8%**

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A 27 week pregnant woman attends her GP with a 12 hour history of a rash. On examination, she has lesions consistent with a diagnosis of chickenpox. Her clinical observations are stable and she is otherwise well.

What would be the recommended management?

- No treatment needed
- Symptomatic treatment only (antihistamine & calamine lotion)
- Oral aciclovir
- Varicella-zoster immunoglobulin (VZIG)
- Oral aciclovir & VZIG

Submit answer

Reference ranges

Score: **22%**

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A 27 week pregnant woman attends her GP with a 12 hour history of a rash. On examination, she has lesions consistent with a diagnosis of chickenpox. Her clinical observations are stable and she is otherwise well.

What would be the recommended management?

No treatment needed	2%
Symptomatic treatment only (antihistamine & calamine lotion)	6%
Oral aciclovir	33%
Varicella-zoster immunoglobulin (VZIG)	30%
Oral aciclovir & VZIG	28%

Pregnant women ≥ 20 weeks who develop chickenpox are generally treated with oral aciclovir if they present within 24 hours of the rash

Important for me Less important

The development of chickenpox in a pregnant woman is associated with increased morbidity, including pneumonia, hepatitis and encephalitis. Therefore, pregnant women who develop a chickenpox rash should see their GP immediately.

Oral aciclovir should be prescribed in pregnant women with chickenpox, if they present within 24 hours of the onset of the rash and are 20 weeks gestation or beyond. (Use before 20 weeks can be considered).
Aciclovir reduces the duration of fever and symptomatology of varicella, if commenced within 24 hours of developing the rash. Symptomatic treatment can be used alongside aciclovir.

Pregnant women with severe chickenpox should be referred to hospital for intravenous aciclovir. In addition, a referral to fetal medicine would be necessary (in the above scenario), due to the small risk of fetal varicella syndrome in the first 28 weeks of pregnancy.

It is important to advise anyone with chickenpox to avoid contact with other potentially susceptible people, such a pregnant women and neonates until the last lesions have crusted over (usually 5 days after the onset of the rash).

VZIG has no therapeutic benefit once the rash has started. RCOG Greentop guidelines, Chickepox in pregnancy.

Discuss (10) | Improve

Next question >

Chickenpox exposure in pregnancy

Chickenpox is caused by primary infection with varicella-zoster virus. Shingles is caused by the reactivation of dormant virus in dorsal root ganglion. In pregnancy, there is a risk to both the mother and also the fetus, a syndrome now termed fetal varicella syndrome

Risks to the mother

- 5 times greater risk of pneumonitis

Fetal varicella syndrome (FVS)

- risk of FVS following maternal varicella exposure is around 1% if occurs before 20 weeks gestation
- studies have shown a very small number of cases occurring between 20-28 weeks gestation and none following 28 weeks
- features of FVS include skin scarring, eye defects (microphthalmia), limb hypoplasia, microcephaly and learning disabilities

Other risks to the fetus

- shingles in infancy: 1-2% risk if maternal exposure in the second or third trimester
- severe neonatal varicella: if the mother develops rash between 5 days before and 2 days after birth there is a risk of neonatal varicella, which may be fatal to the newborn child in around 20% of cases

Management of chickenpox *exposure* in pregnancy, i.e. post-exposure prophylaxis (PEP)

- if there is any doubt about the mother previously having chickenpox maternal blood should be urgently checked for varicella antibodies
- historically, exposure has been managed through the timely administration of varicella zoster immunoglobulin (VZIG). However, the guidance has changed due to a national/international VZIG shortage. This was initially a short-term deviation from practice in 2022 but has now become baked into longer-term guidance
- oral aciclovir (or valaciclovir) is now the first choice of PEP for pregnant women at any stage of pregnancy
    - antivirals should be given at day 7 to day 14 after exposure, not immediately

- why wait until days 7-14? From the PHE guidelines: *'In a study evaluating the comparative effectiveness of 7 days course of aciclovir given either immediately after exposure or starting at day 7 after exposure to healthy children, the incidence and severity of varicella infection was significantly higher in those given aciclovir immediately (10/13 (77%) who received aciclovir immediately developed clinical varicella compared with 3/14 (21%) who started aciclovir at day 7)'*

Management of chickenpox in pregnancy

- if a pregnant woman develops chickenpox in pregnancy then specialist advice should be sought
- there is an increased risk of serious chickenpox infection (i.e. maternal risk) and fetal varicella risk (i.e. fetal risk) balanced against theoretical concerns about the safety of aciclovir in pregnancy
- consensus guidelines (Health Protection Authority and RCOG) suggest **oral aciclovir** should be given if the pregnant women is ≥ 20 weeks and she presents within 24 hours of onset of the rash
- if the woman is < 20 weeks the aciclovir should be 'considered with caution'



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Greenbook 14 4

Varicella

RCOG 18 3

Chickenpox in pregnancy guidelines

UK Health Security Agency 6 1

Guidelines on post exposure prophylaxis (PEP) for varicella or shingles (January 2023)

Suggest link Report broken link

Media



Varicella zoster virus

Osmosis - YouTube 0 0

Report broken media

Score: **16.8%**

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A 20-year-old law student had been backpacking around India for 2 months over her summer holiday. She spent time in a number of rural and coastal areas and volunteered at a wildlife conservation project for 2 weeks. She took malaria prophylaxis throughout.

A few days before her return to the UK she noticed a small nodule on her left forearm. The nodule has gradually enlarged and ulcerated over the last 4 weeks to form a 4cm painless ulcer.

What is the most appropriate investigation to confirm the diagnosis?

- Biopsy of a draining lymph node
- Montenegro skin test
- Punch biopsy of the lesion for histology
- Serology
- Serum PCR

Submit answer

Reference ranges

Score: **22%**

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A few days before her return to the UK she noticed a small nodule on her left forearm. The nodule has gradually enlarged and ulcerated over the last 4 weeks to form a 4cm painless ulcer.

What is the most appropriate investigation to confirm the diagnosis?

Biopsy of a draining lymph node	3%
Montenegro skin test	5%
Punch biopsy of the lesion for histology	72%
Serology	15%
Serum PCR	6%

Cutaneous leishmaniasis is typically diagnosed using a punch biopsy

Important for me Less important

This patient has cutaneous leishmaniasis. This is a protozoal infection mainly caused by *Leishmania major*, *Leishmania tropica* and *Leishmania aethiopica* in Asia, Africa, the Middle East and the Mediterranean, and by *Leishmania braziliensis* and *Leishmania mexicana* in Central and South America. It presents classically with a slowly developing painless ulcer at the site of a sandfly bite.

The most appropriate investigation to confirm the diagnosis is **punch biopsy of the lesion for histology**. The biopsy should be taken from the outer border of the ulcer and should be full thickness. The specimen can be examined using Wright or Giemsa stains to look for amastigotes in the tissue. Culture or PCR can also be performed on the tissue sample.

Biopsy of a draining lymph node is incorrect. You are unlikely to see parasites in tissue samples outside of the ulcer itself in cutaneous leishmaniasis, as the infection is limited to the ulcer only.

Montenegro skin test is incorrect. This is a test in which killed promastigotes are injected under the skin, and the degree of induration at the site is read 48-72 hours later. It is used in some South

American countries but is not recommended as the sensitivity is low and it cannot differentiate between past and current infections.

Serology is incorrect. This is not used in diagnosing cutaneous leishmaniasis because antibody levels are generally low, meaning that the sensitivity of the test is low. The specificity is also variable and there may be cross-reactivity with other infections.

Serum PCR is incorrect. PCR can be carried out on tissue samples from the lesion but is not carried out on serum as there would be minimal or no protozoal DNA in the circulating blood, so the sensitivity would be too low.

Discuss (3) Improve

Next question

Leishmaniasis

Leishmaniasis is caused by the intracellular protozoa *Leishmania*, which are spread by the bites of sandflies. Cutaneous, mucocutaneous leishmaniasis and visceral forms are seen

Cutaneous leishmaniasis

- caused by *Leishmania tropica* or *Leishmania mexicana*
- crusted lesion at the site of bite
- there may be an underlying ulcer
- it is typically diagnosed by doing a punch biopsy from the edge of the lesion allowing for both histology and culture
- cutaneous leishmaniasis acquired in South or Central America merits treatment due to the risk of mucocutaneous leishmaniasis whereas disease acquired in Africa or India can be managed more conservatively





Mucocutaneous leishmaniasis

- caused by *Leishmania braziliensis*
- skin lesions may spread to involve mucosae of nose, pharynx etc

Visceral leishmaniasis (kala-azar)

- mostly caused by *Leishmania donovani*
- occurs in the Mediterranean, Asia, South America, Africa
- fever, sweats, rigors
- massive splenomegaly. hepatomegaly
- poor appetite*, weight loss
    - occasionally patients may report increased appetite with paradoxical weight loss
- grey skin - 'kala-azar' means black sickness
- pancytopaenia secondary to hypersplenism
- the gold standard for diagnosis is bone marrow or splenic aspirate
- management
    - the BNF recommends sodium stibogluconate, an organic pentavalent antimony compound
    - amphotericin B may be used with or after treatment with an antimony compound



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 9 8

2011 Leishmaniasis review

DermNet NZ 9 6

Leishmaniasis review

Suggest link Report broken link

Media



What is Leishmaniasis? An introduction and overview

Let's Learn About Bugs - YouTube 5 2

Report broken media

Score: **16.8%**

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A 43-year-old man presents to the emergency department with a fever, rash, and headache. He has recently returned from travelling around the Solomon Islands. He has no past medical history of note.

On examination, he has a diffuse morbilliform rash and a dark, scab-like lesion is noted on his leg. His temperature is 38.4ºC.

Given the likely diagnosis, what is the most appropriate management?

- Atovaquone-proguanil
- Ceftriaxone
- Ciprofloxacin
- Doxycycline
- Supportive management

Submit answer

Reference ranges

Score: **22%**

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On examination, he has a diffuse morbilliform rash and a dark, scab-like lesion is noted on his leg. His temperature is 38.4ºC.

Given the likely diagnosis, what is the most appropriate management?

Atovaquone-proguanil	5%
Ceftriaxone	5%
Ciprofloxacin	10%
Doxycycline	73%
Supportive management	7%

Doxycycline is typically used in the management of typhus

Important for me Less important

Doxycycline is correct. The patient presents with a fever, headache, and rash. These are common features of many illnesses in the returning traveller. However, the dark scab-like lesion is more discriminating and in the context of a patient returning from the Solomon Islands, this likely represents the black eschar associated with scrub typhus infection. The Solomon Islands from the southern tip of the 'Tsutsugamushi Triangle' where scrub typhus is endemic. The treatment is with doxycycline.

Atovaquone-proguanil is incorrect. Also known as Malarone, this is an accepted oral treatment for uncomplicated malaria. Malaria is present in the Solomon Islands but a rash is actually uncommon in malaria and a black eschar is not a feature.

Ceftriaxone is incorrect. This would be an appropriate treatment for typhoid, which is a common cause of fever in returning travellers. A black eschar is not a feature of typhoid infection.

Ciprofloxacin is incorrect. This would be an appropriate treatment if we suspected anthrax. This is

another cause of eschar. However, while a patient with anthrax may be systemically unwell, a diffuse morbilliform rash is not typical. Additionally, the eschar of anthrax may be associated with some perilesional oedema and blister which are not mentioned here.

Supportive management is incorrect. The mortality rate of scrub typhus is 6% untreated. Management is with doxycycline.

Discuss | Improve

Next question >

Typhus

Typhus is a group of rickettsial diseases transmitted between hosts by arthropods. Examples include

- endemic typhus
  - caused by *Rickettsia typhi*
  - reservoir/vector: fleas on rats
  - occurs throughout the world, typically in warm costal regions
  - relative bradycardia
- epidemic typhus
  - caused by *Rickettsia prowazekii*
  - reservoir/vector: body louse
  - more common in central and eastern Africa, central and South America
- scrub typhus
  - caused by *Orientia tsutsugamushi*
  - reservoir/vector: harvest mites on humans or rodents
  - more common in Asia
  - black eschar at site of original inoculation
  - relative bradycardia despite fever
- spotted fever
  - caused by Rickettsia spotted fever group
  - spread by ticks
  - examples include Rocky Mountain spotted fever

Common features

- fever, headache, malaise
- rash
  - typically maculopapular
  - begins on the trunk and spreads to the extremities
- later complications
  - meningoencephalitis

Management

- doxycycline

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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A 46-year-old man telephones his GP for advice. Five days ago, he was in contact with his nephew, who was unwell and had a rash at the time. It has since been confirmed that his nephew has measles. The patient is concerned as he was not immunised against measles as a child. Furthermore, he takes methotrexate for rheumatoid arthritis.

He has no symptoms currently.

What is the most appropriate treatment?

- Immunoglobulin
- Measles, mumps and rubella (MMR) vaccine
- Measles, mumps and rubella (MMR) vaccine and immunoglobulin
- Measles-only vaccine
- No treatment

Submit answer

Reference ranges

Score: **22%**

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He has no symptoms currently.

What is the most appropriate treatment?

Immunoglobulin	31%
Measles, mumps and rubella (MMR) vaccine	15%
Measles, mumps and rubella (MMR) vaccine and immunoglobulin	38%
Measles-only vaccine	4%
No treatment	12%

Those exposed to measles can be given immunoglobulin to reduce the risk of infection

Important for me Less important

The most appropriate course of action, for a patient who is immunosuppressed and does not have adequate immunity to measles, is to administer **immunoglobulin** following exposure. Ideally, this should be done within 72 hours, but can be done up until 6 days post-exposure.

For patients with normal immunity, who have not been fully immunised against measles, the **MMR vaccine** should be administered within 72 hours of exposure.

Administering both the **MMR vaccine and immunoglobulin** is inappropriate. This can invalidate the effect of the vaccine.

Giving the **measles-only vaccine** would also be inappropriate. This patient should be given immunoglobulin. For a patient with normal immunity, who is not fully immunised, the MMR vaccine should be given rather than a measles-only vaccine.

No treatment is incorrect. Given this patient's clear exposure and history of both immunosuppression and lack of immunisation against measles, immunoglobulin should be given.

Measles

Measles is now rarely seen in the developed world following the adoption of immunisation programmes. Outbreaks are occasionally seen, particularly when vaccinations rates drop, for example after the MMR controversy of the early 2000s.

Overview

- RNA paramyxovirus
- one of the most infectious known viruses
- spread by aerosol transmission
- infective from prodrome until 4 days after rash starts
- incubation period = 10-14 days

Features

- prodromal phase
  - irritable
  - conjunctivitis
  - fever
- Koplik spots
  - typically develop before the rash
  - white spots ('grain of salt') on the buccal mucosa
- rash
  - starts behind ears then to the whole body
  - discrete maculopapular rash becoming blotchy & confluent
  - desquamation that typically spares the palms and soles may occur after a week
- diarrhoea occurs in around 10% of patients



© Image used on license from DermNet NZ

Koplik spots

Investigations

- IgM antibodies can be detected within a few days of rash onset

Management

- mainly supportive
- admission may be considered in immunosuppressed or pregnant patients
- notifiable disease → inform public health

Complications

- otitis media: the most common complication
- pneumonia: the most common cause of death
- encephalitis: typically occurs 1-2 weeks following the onset of the illness)
- subacute sclerosing panencephalitis: very rare, may present 5-10 years following the illness
- febrile convulsions
- keratoconjunctivitis, corneal ulceration
- diarrhoea
- increased incidence of appendicitis
- myocarditis



© Image used on license from DermNet NZ

The rash typically starts behind the ears and then spreads to the whole body

Management of contacts

- if a child not immunized against measles comes into contact with measles then MMR should be offered (vaccine-induced measles antibody develops more rapidly than that following natural infection)
- this should be given within 72 hours

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 5 6

Subacute sclerosing panencephalitis

Suggest link Report broken link

Media



Measles - What is it?

Let's Learn About Bugs - YouTube 3 0



Measles: Features, Management and Mnemonics

Townsend Teaching - YouTube 2 0



Measles

Osmosis - YouTube 4 1

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A 35-year-old man is referred to a tuberculosis (TB) clinic after his wife is confirmed to have fully sensitive pulmonary TB. There is no history of cough, fevers or weight loss. He has no medical problems, does not take any medication regularly and does not smoke or drink alcohol. His blood tests show:

HIV antibody test	negative
Hepatitis B surface antigen test	negative
Hepatitis B surface antibody test	positive
Hepatitis C antibody test	negative
Interferon-gamma release antibody	positive

What is the most appropriate treatment to offer initially?

- 2 months of rifampicin, isoniazid, pyrazinamide, ethambutol and pyridoxine
- 2 months of tenofovir
- 3 months of rifampicin, isoniazid and pyridoxine
- 48 weeks of peginterferon alfa-2a
- 6 months of rifampicin only

Submit answer

Reference ranges

Score: **22%**

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HIV antibody test	negative
Hepatitis B surface antigen test	negative
Hepatitis B surface antibody test	positive
Hepatitis C antibody test	negative
Interferon-gamma release antibody	positive

What is the most appropriate treatment to offer initially?

2 months of rifampicin, isoniazid, pyrazinamide, ethambutol and pyridoxine	23%
2 months of tenofovir	2%
3 months of rifampicin, isoniazid and pyridoxine	56%
48 weeks of peginterferon alfa-2a	3%
6 months of rifampicin only	17%

Latent tuberculosis treatment options:

- 3 months of isoniazid (with pyridoxine) and rifampicin, or
- 6 months of isoniazid (with pyridoxine)

Important for me Less important

AI

This patient has latent TB as evidenced by a positive interferon-gamma release antibody (IGRA) test and no other symptoms consistent with active TB. NICE advises that treatment for latent TB should be with either **3 months of rifampicin, isoniazid and pyridoxine** or 6 months of isoniazid and pyridoxine.

This patient has either been immunised against Hepatitis B or previously infected and therefore has a positive Hepatitis B surface antibody test but a negative antigen test. If they had chronic

hepatitis B they would have a positive antigen test. Treatment of chronic hepatitis B includes once-weekly injections of **peginterferon alfa-2a, a recombinant interferon, for 48 weeks** or antivirals such as **tenofovir** or entecavir.

If this patient had a positive IGRA test and symptoms consistent with active TB, they would require treatment for active TB with **2 months of rifampicin, isoniazid, pyrazinamide, ethambutol and pyridoxine** followed by 4 months of rifampicin, isoniazid and pyridoxine.

6 months of rifampicin only can be used to treat patients with latent TB but is not recommended by NICE guidelines. It could also be used if this patient's wife had isoniazid-resistant pulmonary TB.

👍 👎 💬 Discuss (3) Improve

Next question ›

Tuberculosis: latent disease ★

Patients with latent tuberculosis are asymptomatic and non-infectious. They are typically diagnosed by having a positive tuberculin skin test or Interferon-Gamma Release Assay (IGRA) combined with a normal chest x-ray, helping to exclude active tuberculosis.

NICE now give two choices for treating **latent tuberculosis**:

- 3 months of isoniazid (with pyridoxine) and rifampicin, or
- 6 months of isoniazid (with pyridoxine)

Why two choices? NICE (2016) state the following:

> *Base the choice of regimen on the person's clinical circumstances. Offer:*
>
> - *3 months of isoniazid (with pyridoxine) and rifampicin to people younger than 35 years if hepatotoxicity is a concern after an assessment of both liver function (including transaminase levels) and risk factors*
> - *6 months of isoniazid (with pyridoxine) if interactions with rifamycins are a concern, for example, in people with HIV or who have had a transplant.*

People with latent tuberculosis cannot pass the disease on to others, so there is no restriction in terms of employment etc. This advice would obviously change if the patient developed active tuberculosis at any point.

Risk factors for developing active tuberculosis include:

- silicosis
- chronic renal failure
- HIV positive
- solid organ transplantation with immunosuppression
- intravenous drug use
- haematological malignancy
- anti-TNF treatment
- previous gastrectomy

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2016 Tuberculosis guidelines

1 0

Greenbook

Tuberculosis

0 0

Suggest link

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Media



Tuberculosis

Osmosis - YouTube 2 0

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A 38-year-old man presents to the emergency department with fever and shivering, occurring approximately every two days. He has returned from India three weeks ago, having spent four months travelling. While he was in India, he was treated for malaria with chloroquine. He has no other past medical history and does not take any regular medications.

Observations:

- Heart rate 88 beats/min
- Blood pressure 120/77 mmHg
- Respiratory rate 18 breaths/min
- Oxygen saturations 98% on room air
- Temperature 38.6°C

On examination, the patient is diaphoretic. There is palpable splenomegaly.

A thick and thin blood film confirms malaria.

What organism is most likely to be responsible?

- *Plasmodium falciparum*
- *Plasmodium knowlesi*
- *Plasmodium malariae*
- *Plasmodium ovale*
- *Plasmodium vivax*

Submit answer

Reference ranges

Score: **22%**

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- Respiratory rate 18 breaths/min
- Oxygen saturations 98% on room air
- Temperature 38.6ºC

On examination, the patient is diaphoretic. There is palpable splenomegaly.

A thick and thin blood film confirms malaria.

What organism is most likely to be responsible?

Plasmodium falciparum	28%
Plasmodium knowlesi	6%
Plasmodium malariae	10%
Plasmodium ovale	13%
Plasmodium vivax	42%

P. vivax and *ovale* have hypnozoite forms, which can cause clinical disease long after patients leave the endemic area or may cause relapsing disease following treatment

Important for me Less important

Plasmodium vivax is the correct answer. The patient has a confirmed diagnosis of malaria, having previously been treated for malaria within the last six months. This suggests a relapse of disease. The two forms of malaria that cause relapsing disease are *Plasmodium vivax* and *Plasmodium ovale*. *Plasmodium vivax* is more common in India while *Plasmodium ovale* is more common in Africa. Chloroquine is the typical treatment in most countries. This medication acts on blood-stage parasites only and does not act on hypnozoites. The only available drug that can kill the hypnozoites is primaquine.

Plasmodium falciparum is incorrect. This type of malaria does not cause relapsing disease.

Plasmodium knowlesi is incorrect. This form of malaria, which is common in South-East Asia does not cause relapsing disease.

Plasmodium malariae is incorrect. This does not cause relapsing disease and is a less common form of malaria. Additionally, it causes fever every three days rather than every two days as in other forms of malaria.

Plasmodium ovale is incorrect. This can also cause relapsing disease. However, it does so far less commonly than *vivax* malaria and is more common in Africa rather than Asia.

👍 👎 💬 Discuss (6) Improve

Next question ›

Malaria: non-falciparum ★

The most common cause of non-falciparum malaria is *Plasmodium vivax*, with *Plasmodium ovale* and *Plasmodium malariae* accounting for the other cases. *Plasmodium vivax* is often found in Central America and the Indian Subcontinent whilst *Plasmodium ovale* typically comes from Africa.

Plasmodium knowlesi is another non-falciparum species which causes clinical pathology, found predominantly in South East Asia.

Features

- general features of malaria: fever, headache, splenomegaly
- *Plasmodium vivax/ovale*: cyclical fever every 48 hours. *Plasmodium malariae*: cyclical fever every 72 hours
- *Plasmodium malariae*: is associated with nephrotic syndrome.

Ovale and vivax malaria have a hypnozoite stage and may therefore relapse following treatment.

Treatment

- in areas which are known to be chloroquine-sensitive then WHO recommend either an artemisinin-based combination therapy (ACT) or chloroquine
- in areas which are known to be chloroquine-resistant an ACT should be used
- ACTs should be avoided in pregnant women
- patients with ovale or vivax malaria should be given primaquine following acute treatment with chloroquine to destroy liver hypnozoites and prevent relapse

Next question

Textbooks

High-yield textbook

Extended textbook

Links

WHO 9 1

Malaria treatment guidelines

Suggest link Report broken link

Media



Malaria

Osmosis - YouTube 4 0

Report broken media

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A 42-year-old woman visits the emergency department with a 4-day history of high fevers and myalgia. She has just landed in the UK from the Democratic Republic of the Congo. Her past medical history includes uterine fibroids and iron deficiency anaemia for which she takes regular ferrous sulfate.

Her observations are as follows:

- Temperature 39.1ºC
- Heart rate 122bpm
- Blood pressure 105/80mmHg
- Respiratory rate 15 breaths/min
- Oxygen saturations 99% on air

On examination, she is clammy to touch and complains of a headache. Her chest sounds are clear and her heart sounds are normal. She has a soft and non-tender abdomen. On closer examination, there are multiple enlarged and tender lymph nodes in her axillae and groin.

What is the most likely causative organism?

- *Burkholderia pseudomallei*
- *Mycobacterium leprae*
- *Treponema pallidum pertenue*
- *Yersinia pestis*
- Zaire ebolavirus

Submit answer

Reference ranges

Score: **22%**

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- Oxygen saturations 99% on air

On examination, she is clammy to touch and complains of a headache. Her chest sounds are clear and her heart sounds are normal. She has a soft and non-tender abdomen. On closer examination, there are multiple enlarged and tender lymph nodes in her axillae and groin.

What is the most likely causative organism?

Burkholderia pseudomallei	19%
Mycobacterium leprae	4%
Treponema pallidum pertenue	11%
Yersinia pestis	42%
Zaire ebolavirus	24%

The plague is caused by the gram-negative bacterium *Yersinia pestis*. Three clinical presentations can occur: bubonic (most common), pneumonic and septicaemic

Important for me Less important

This patient has a diagnosis of the bubonic plague, caused by the gram-negative bacterium ***Yersinia pestis***. It is transmitted by fleas and is endemic to the Democratic Republic of the Congo, Madagascar, and Peru. Symptoms typically include flu-like symptoms from 3-7 days post-exposure followed by the eruption of inflamed, tense and painful lymph nodes. The disease carries a significantly high mortality rate if not treated and prompt recognition and antibiotic administration (i.e. Streptomycin) can reduce the mortality from 60% to 15%.

Melioidosis is a bacterial illness caused by ***Burkholderia pseudomallei***. It is a tropical disease seen in South-East Asia, Africa, the Middle East and the South Pacific. Some may present with a localised skin infection from exposure to the bacterium as it enters the skin (e.g. nodule or ulcer formation) whereas other patient's progress to septicaemia and abscess formation. Melioidosis is not associated with inflamed and painful lymph nodes making it an unlikely diagnosis.

Leprosy is a granulomatous disease caused by ***Mycobacterium leprae*** which affects the peripheral nerves and skin causing symptoms such as loss of temperature and light touch followed by loss of pain sensation. Associated cutaneous lesions follow later in the disease and appear as hypopigmented patches of skin. These symptoms are not seen in the above patient and leprosy is not associated with painful lymphadenopathy.

Yaws is a chronic skin infection caused by the bacterium ***Treponema pallidum pertenue***. There are several stages of illness with the disease initially presenting as a papule from the site of infection that later ulcerates. As the disease progresses, more widespread papules appear over the rest of the body. These lesions are not seen with this patient, making Yaws incorrect.

Ebola virus disease is an extremely deadly disease caused by multiple different viruses including **Zaire ebolavirus**. The disease causes flu-like symptoms including fever, myalgia and sore throat along with gastrointestinal symptoms such as abdominal pain and vomiting. Zaire ebolavirus causes a haemorrhagic illness and as the disease progresses, patients develop unexplained bleeding and bruising. Although this patient has flown from a high-risk area for the Ebola virus, she has no gastrointestinal symptoms or haemorrhagic symptoms.

Discuss (1) | Improve

Next question >

Bubonic plague ★

Bubonic plague is the commonest form of plague in humans. Fleas transmit the bacteria from rodents to humans via their bite. The bacteria can also be spread from one infected human to another if it develops into pneumonic plague (in the lungs) via aerosolized particles.

Bubonic plague is still endemic in many countries. *Yersinia pestis* is recognised as the causative organism.

Typically flu-like symptoms develop 3-7 days after exposure; High temperature, headache, weakness. The lymph nodes exposed then becomes inflamed, tense and painful.

Treatment reduces mortality from 60% to 15% and is typically with antibiotics such as streptomycin.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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A 38 year-old woman presented to the medical outpatient clinic with an 8 month history of deteriorating vision. She also complained of a pruritic rash intermittently affecting her forearms and neck. She had recently migrated to the UK from Guinea, where she had lived since birth. She had suffered from malaria as a child but had been fit and well since. She did not smoke and drank 6-10 units of alcohol per week.

On examination, her temperature was 36.7ºC, pulse was 68 beats per minute and blood pressure was 124/80 mmHg. Her chest was clear on auscultation and heart sounds were normal. Fundoscopy was not possible due to clouding of both corneas. There was evidence of a mottled rash over the forearms and neck with a leopard print appearance.

Investigations:

Haemoglobin	141 g/L (130-180)
White cell count	6.7 x ^{9}/L (4.0-11.0)
Neutrophil count	3.0 x ^{9}/L (2.0-7.5)
Lymphocyte count	1.7 x ^{9}/L (1.3-3.5)
Eosinophil count	0.6 X ^{9}/L (0.1-0.4)
Platelets	260 x ^{9}/L (150-400)

Sodium	138 mmol/L (135-145)
Potassium	4.3 mmol/L (3.5-5.0)
Urea	7.2 mmol/L (2.5-7.5)
Creatinine	61 mol/L (25-95)
Fasting plasma glucose	5.0 mmol/L (3.0-6.0)

Giemsa-stained blood film	No abnormality detected

Given the likely diagnosis, what is the most appropriate treatment?

- Diethylcarbamazine (DEC)
- Ivermectin
- Benzinidazole

Albendazole

Praziquantel

Submit answer

Reference ranges

Score: **22%**

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A 38 year-old woman presented to the medical outpatient clinic with an 8 month history of deteriorating vision. She also complained of a pruritic rash intermittently affecting her forearms and neck. She had recently migrated to the UK from Guinea, where she had lived since birth. She had suffered from malaria as a child but had been fit and well since. She did not smoke and drank 6-10 units of alcohol per week.

On examination, her temperature was 36.7ºC, pulse was 68 beats per minute and blood pressure was 124/80 mmHg. Her chest was clear on auscultation and heart sounds were normal. Fundoscopy was not possible due to clouding of both corneas. There was evidence of a mottled rash over the forearms and neck with a leopard print appearance.

Investigations:

Haemoglobin	141 g/L (130-180)
White cell count	6.7 x ^{9}/L (4.0-11.0)
Neutrophil count	3.0 x ^{9}/L (2.0-7.5)
Lymphocyte count	1.7 x ^{9}/L (1.3-3.5)
Eosinophil count	0.6 X ^{9}/L (0.1-0.4)
Platelets	260 x ^{9}/L (150-400)

Sodium	138 mmol/L (135-145)
Potassium	4.3 mmol/L (3.5-5.0)
Urea	7.2 mmol/L (2.5-7.5)
Creatinine	61 mol/L (25-95)
Fasting plasma glucose	5.0 mmol/L (3.0-6.0)

Giemsa-stained blood film	No abnormality detected

Given the likely diagnosis, what is the most appropriate treatment?

Diethylcarbamazine (DEC)	29%
Ivermectin	44%
Benzinidazole	5%

Albendazole 11%

Praziquantel 10%

This patient has presented with features of onchocerciasis, a disease caused by the filarial nematode Onchocerca volvulus. The parasite is transmitted by blackflies of the genus Simulium, which deposit the parasites larva onto the skin when biting to extract blood. The adult worm lives in subcutaneous nodules and produces larvae, which become deposited in the skin and eyes. Disease manifests when the parasite dies in tissues and releases Wolbachia endosymbiotic bacteria into tissues, provoking a host immune response. This above patients disease has manifested as river blindness, where the worms die in the cornea and provoke chronic inflammation, eventually leading to clouding of the cornea. Inflammation in subcutaneous tissues leads to a pruritic rash and formation of a leopard-skin appearance over time. The worms can be visualised by microscopy of skin snip biopsy. The treatment of choice is ivermectin; DEC should not be used because rapid death of the worms can exacerbate damage to surround tissues and even cause complete blindness.

Discuss (2) Improve

Next question

Helminths

Nematodes (roundworms)

Worm	Notes	Treatment
Strongyloides stercoralis	Larvae are present in soil and gain access to the body by penetrating the skin Features include diarrhoea, abdominal pain, papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks, larva currens: pruritic, linear, urticarial rash, if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered	Ivermectin and -bendazoles are used
Enterobius vermicularis (pinworm)	Threadworm infestation is asymptomatic in around 90% of cases, possible features include perianal itching, particularly at night; girls may have vulval symptoms	-bendazoles

Worm	Notes	Treatment
	Diagnosis may be made by the applying sticky plastic tape to the perianal area and sending it to the laboratory for microscopy to see the eggs	
Ancylostoma duodenale, *Necator americanus* (hookworms)	Larvae penetrate skin of feet; gastrointestinal infection → anaemia Thin-shelled ova	-bendazoles
Loa loa	Transmission by deer fly and mango fly Causes red itchy swellings below the skin called 'Calabar swellings', may be observed when crossing conjunctivae	Diethylcarbamazine
Trichinella spiralis	Typically develops after eating raw pork Features include fever, periorbital oedema and myositis (larvae encyst in muscle)	-bendazoles
Onchocerca volvulus	Causes 'river blindness'. Spread by female blackflies Features include blindness, hyperpigmented skin and possible allergic reaction to microfilaria	Ivermectin r**IVER**blindness = **IVER**mectin
Wuchereria bancrofti	Transmission by female mosquito Causes blockage of lymphatics → elephantiasis	Diethylcarbamazine
Toxocara canis (dog roundworm)	Transmitted through ingestion of infective eggs. Features include visceral larva migrans and retinal granulomas VISCious dogs → blindness	Diethylcarbamazine
Ascaris lumbricoides (giant roundworm)	Eggs are visible in faeces May cause intestinal obstruction and occasional migrate to lung (Loffler's syndrome)	-bendazoles

Cestodes (tapeworms)

Worm	Notes	Treatment
Echinococcus granulosus	Transmission through ingestion of eggs in dog faeces. Definite host is dog, which ingests hydatid cysts from sheep, who act as an intermediate host. Often seen in farmers. Features include liver cysts and anaphylaxis if cyst ruptures (e.g. during surgical removal)	- bendazoles
Taenia solium	Often transmitted after eating undercooked pork. Causes cysticercosis and neurocysticercosis, mass lesions in the brain 'swiss cheese appearance'	- bendazoles

Trematodes (flukes)

Worm	Notes	Treatment
Schistosoma haematobium	Hosted by snails, which release cercariae that penetrate skin. Causes 'swimmer's itch' - frequency, haematuria. Risk factor for squamous cell bladder cancer	Praziquantel
Paragonimus westermani	Caused by undercooked crabmeat, results in secondary bacterial infection of lungs	Praziquantel
Clonorchis sinensis	Caused by undercooked fish Features include biliary tract inflammation. Known risk factor for cholangiocarcinoma	Praziquantel
Fasciola hepatica (the liver fluke)	May cause biliary obstruction	Triclabendazole



Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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Back to top

A 29-year-old lady comes to see you for advice having seen news of the recent Zika virus outbreak. She and her husband are planning on starting a family, but she has only arrived back from Brazil last week after a business trip. She has not experienced any fever or worrying symptoms, either during travel or since arrival back in the UK. What would be the most appropriate advice to give her?

- Start 5mg folic acid daily
- If no fever can become pregnant
- Need to check Zika virus serology before becoming pregnant
- Should avoid becoming pregnant for at least 2 weeks after travel
- Should avoid becoming pregnant for at least 8 weeks after travel

Submit answer

Reference ranges

Score: **22%**

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Start 5mg folic acid daily	14%
If no fever can become pregnant	6%
Need to check Zika virus serology before becoming pregnant	23%
Should avoid becoming pregnant for at least 2 weeks after travel	17%
Should avoid becoming pregnant for at least 8 weeks after travel	**39%**

The latest advice as per Public Health England is that women who are considering becoming pregnant should avoid becoming pregnant while travelling in an area with active Zika virus transmission, and for 8 weeks after their return.

Whilst folic acid is recommended to women trying for pregnancy, the usual dose is 400 micrograms daily and there is currently no recommendation to increase this after Zika exposure. The test for Zika serology is only recommended for patients with a relevant travel history and current symptoms.

Discuss (5) | Improve

Next question >

Zika virus

Zika is a mosquito-borne infection caused by Zika virus, a member of the genus flavivirus and family Flaviviridae. It was first isolated from a monkey in the Zika forest in Uganda in 1947.

Transmission is usually via the bite of an infected Aedes mosquito, although a small number of cases of sexual transmission have been reported. There is increasing evidence of transmission via the placenta from mother to fetus.

The majority of people infected with Zika virus have no symptoms. For those with symptoms, Zika virus tends to cause a mild, short-lived (2 to 7 days) febrile disease. Signs and symptoms suggestive of Zika virus infection may include a combination of the following:

- fever
- rash
- arthralgia/arthritis
- conjunctivitis
- myalgia
- headache
- retro-orbital pain
- pruritus

Serious complications in adults are not common, although the virus has been associated with Guillain-Barre syndrome. Scientific consensus however has linked Zika with microcephaly and other congenital abnormalities, which has led the World Health Organisation (WHO) to declare a Public Health Emergency of International Concern (PHEIC).

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Public Health England

Zika virus guidance

0 1

Suggest link Report broken link

Media



Zika virus

Osmosis - YouTube 1 0

Report broken media

Score: **16.8%**

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A 27-year-old man who recently immigrated to the UK from Ethiopia complains of a chronic cough with night sweats for five weeks. On direct questioning, he also admits coughing up small volumes of bright red blood a few times. This has made him especially concerned as he his father died from lung related problems following chronic coughs.

Following a chest X-ray and blood tests he is diagnosed with TB and contact tracing is started. His 25-year-old male partner lives with him and is identified as being at high risk of contracting TB. The partner undergoes Mantoux testing and has a 2mm area of induration. He has never had BCG vaccination as far as he is aware and has no vaccination scar. What is the most appropriate management that should be offered to the partner?

- BCG vaccination
- HIV testing and if negative then BCG vaccination
- Prophylactic anti-TB treatment
- Repeat TB screening in six weeks
- No further management needed

Submit answer

Reference ranges

Score: **22%**

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BCG vaccination	17%
HIV testing and if negative then BCG vaccination	42%
Prophylactic anti-TB treatment	25%
Repeat TB screening in six weeks	8%
No further management needed	8%

The correct answer is HIV testing and if negative then BCG vaccination. The low area of response to the Mantoux testing indicates that he is unlikely to have TB and has not been vaccinated. However, the test can be a false negative result in an immunosuppressed patient. As the partner is at increased risk of HIV, NICE advises that an HIV test should be done prior to vaccination. BCG vaccination is a live vaccination and therefore is contraindicated in an immunosuppressed patient. Of note, previous vaccination can lead to a false positive result.

NICE advises that once a diagnosis of pulmonary TB has been made then close contacts should be managed as follow:

- If asymptomatic and younger than 65 years then test for latent TB. If Mantoux-negative and unvaccinated then offer vaccination. If at risk of HIV then test for HIV first.
- If asymptomatic and older than 65 years then assess with a chest X-ray.

Treatment for TB should be considered if TB is confirmed and not used prophylactically. Repeat screening is generally not used.

Source:

'Tuberculosis NICE Guidelines.' The National Institute for Health and Care Excellence, 13 Jan. 2016.

Discuss (7) | Improve

Next question >

Tuberculosis: screening and diagnosis

Screening for latent tuberculosis

The Mantoux test is the main technique used to screen for latent tuberculosis. In recent years the interferon-gamma blood test has also been introduced. It is used in a number of specific situations such as:

- the Mantoux test is positive or equivocal
- people where a tuberculin test may be falsely negative (see below)

Mantoux test

- 0.1 ml of 1:1,000 purified protein derivative (PPD) injected intradermally
- result read 2-3 days later

Diameter of induration	Positivity	Interpretation
< 6mm	Negative - no significant hypersensitivity to tuberculin protein	Previously unvaccinated individuals may be given the BCG
6 - 15mm	Positive - hypersensitive to tuberculin protein	Should not be given BCG. May be due to previous TB infection or BCG
> 15mm	Strongly positive - strongly hypersensitive to tuberculin protein	Suggests tuberculosis infection.

False negative tests may be caused by:

- miliary TB
- sarcoidosis
- HIV
- lymphoma
- very young age (e.g. < 6 months)

Heaf test

The Heaf test was previously used in the UK but has been since been discontinued. It involved injection of PPD equivalent to 100,000 units per ml to the skin over the flexor surface of the left forearm. It was then read 3-10 days later.

Diagnosis of active tuberculosis

Chest x-ray

- upper lobe cavitation is the classical finding of reactivated TB
- bilateral hilar lymphadenopathy

Sputum smear

- 3 specimens are needed
- rapid and inexpensive test
- stained for the presence of acid-fast bacilli (Ziehl-Neelsen stain)
  - all mycobacteria will stain positive (i.e. nontuberculous mycobacteria)
- the sensitivity is between 50-80%
  - this is decreased in individuals with HIV to around 20-30%

Sputum culture

- the gold standard investigation
  - more sensitive than a sputum smear and nucleic acid amplification tests
  - can assess drug sensitivities
- can take 1-3 weeks (if using liquid media, longer if solid media)

Nucleic acid amplification tests (NAAT)

- allows rapid diagnosis (within 24-48 hours)
- more sensitive than smear but less sensitive than culture

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

NICE

7 3

2016 Tuberculosis guidelines

Suggest link

Report broken link

Media



Tuberculosis

Osmosis - YouTube 2 1

Report broken media

Score: **16.8%**

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A 35-year-old man is referred to the medical assessment unit by his general practitioner. He reports two months of episodic swellings of the soft tissue near various joints that are hot and occasionally pruritic. He has no past medical history and is not on any regular medications. He has recently returned from spending six months in Equatorial Guinea.

On examination, there is a large, 2 x 3cm non-pitting swelling of his dorsal forearm that is hot but not erythematous.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	14.2 * 10^9/L	(4.0 - 11.0)
Neuts	6.5 * 10^9/L	(2.0 - 7.0)
Lymphs	2.2 * 10^9/L	(1.0 - 3.5)
Mono	0.3 * 10^9/L	(0.2 - 0.8)
Eosin	5.2 * 10^9/L	(0.0 - 0.4)

What is the likely diagnosis?

- Cutaneous larva migrans
- Cutaneous leishmaniasis
- Loiasis
- Schistosomiasis
- Syphilis

Submit answer

Reference ranges

Score: **22%**

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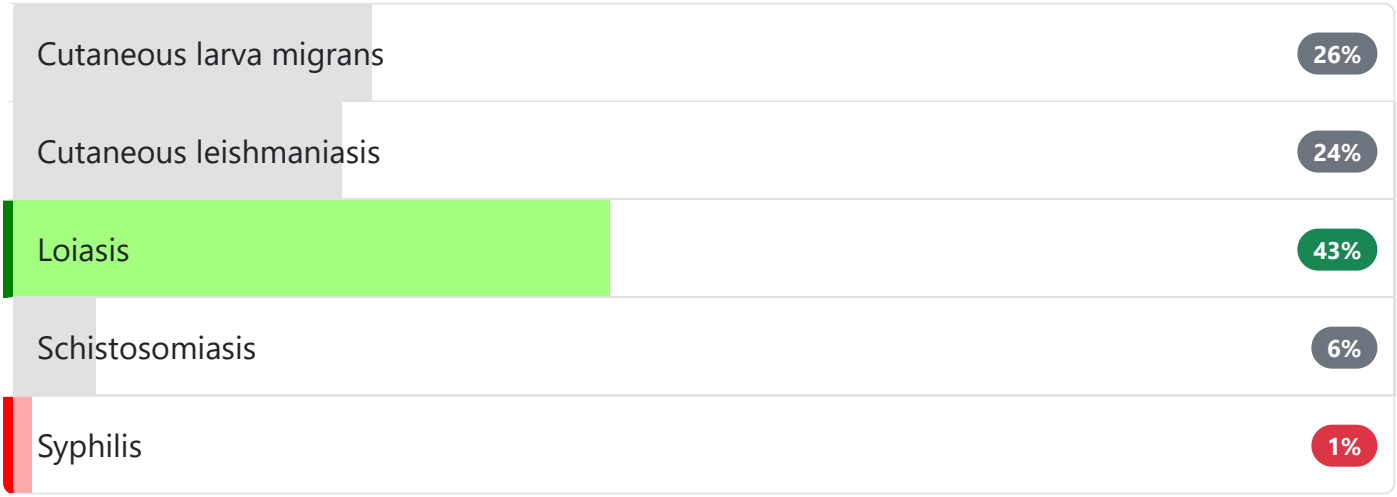
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On examination, there is a large, 2 x 3cm non-pitting swelling of his dorsal forearm that is hot but not erythematous.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	14.2 * 10^9/L	(4.0 - 11.0)
Neuts	6.5 * 10^9/L	(2.0 - 7.0)
Lymphs	2.2 * 10^9/L	(1.0 - 3.5)
Mono	0.3 * 10^9/L	(0.2 - 0.8)
Eosin	5.2 * 10^9/L	(0.0 - 0.4)

What is the likely diagnosis?



Calabar swellings: transient, non-erythematous, hot swelling of soft-tissue around joints → loiasis

Important for me Less important

Loiasis is the correct answer. The patient presents with transient, non-erythematous, hot swellings of the soft tissues around joints. This is a description of so-called Calabar swellings, which are

associated with loiasis. This condition is a filarial infection caused by the nematode Loa Loa. The Calabar swellings are caused by an immune reaction to the worms. Eosinophilia is common in this condition and it is endemic to Equatorial Guinea.

Cutaneous larva migrans is incorrect. This can cause skin lesions and eosinophilia. However, the lesions in this condition (which is caused by a hookworm infection) are classically serpiginous in outline and therefore a 2x3cm lesion would not be characteristic.

Cutaneous leishmaniasis is incorrect. This condition is transmitted by sandflies. It characteristically causes an ulcerative lesion, which is not present in this case as there is no mention of skin erosion. Additionally, the lesions tend not to be transient and persist without treatment.

Schistosomiasis is incorrect. An acute schistosomiasis infection is typically characterized by fever, blotchy rash, myalgia, arthralgia and diarrhoea. So while it is endemic in Equatorial Guinea and associated with blood eosinophilia, the presentation is not characteristic of the disease.

Syphilis is incorrect. This can cause a skin lesion called a chancre, which is a painless ulcer. However, this typically occurs on the genitals and occurs once rather than episodically. Additionally, eosinophilia is not typical of this condition.

Discuss (3) Improve

Next question

Loiasis

Loiasis is a filarial infection caused by Loa Loa. It is transmitted by the Chrysops deerfly and tends to occur in rainforest regions of Western and Central Africa.

Clinical features

- pruritus
- urticaria
- Calabar swellings: transient, non-erythematous, hot swelling of soft-tissue around joints
- 'eye worm' - the dramatic presentation of subconjunctival migration of the adult worm.

It has fewer pathological features than other the microfilarial infections Onchocerciasis and Lymphatic Filariasis. However high loa loa microfilaraemia is associated with encephalopathy following treatment with either Ivermectin or DEC. This occurs due to the death of vast numbers of blood microfilaria. Both of these drugs are contraindicated if loa loa microfilaraemia exceeds 2500 mf/ml.

This has significant public health implications as Ivermectin is currently the drug of choice for

control of both Onchocerciasis and Lymphatic Filariasis in Africa.



Adult Loa loa parasite. Loa loa is the filarial nematode (roundworm) species that causes loa loa filariasis. It is commonly known as the 'eye worm.' Its geographic distribution includes Africa and India. Credit: NIAID

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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Back to top

A 25-year-old man is seen in the walk-in travellers clinic. He returned from a holiday in Brazil 5 days ago and has a 1 day history of fever, headache and myalgia. He is usually fit and well. He tells you he took regular malaria prophylaxis while away.

On examination he has a heart rate of 110 beats per minute and a blood pressure of 102/72 mmHg. His temperature is 38.1 ºC and he has dry mucous membranes. Examination of cardiovascular, respiratory and gastrointestinal systems is unremarkable but he has multiple mosquito bites over his arms and legs.

His blood tests are as follows:

Hb	132 g/l	Na^+	144 mmol/l	Bilirubin	8 µmol/l
Platelets	91 * 10^9/l	K^+	3.5 mmol/l	ALP	98 u/l
WBC	13 * 10^9/l	Urea	9.7 mmol/l	ALT	22 u/l
Neuts	11 * 10^9/l	Creatinine	111 µmol/l	γGT	14 u/l
Lymphs	0.6 * 10^9/l	CRP	78 mg/l	Albumin	40 g/l

He is admitted for broad spectrum antibiotics, fluids and analgesia but his fever continues. His malaria films are negative but he has a positive PCR for dengue virus. Antibiotics are stopped but fluids are continued. His fever and headache settle though he does developed some mild ankle oedema.

Which of the following is true regarding his discharge?

- Dengue shock is unlikely to occur after 24 hours post-fever
- Dengue shock is unlikely to occur once platelets improving
- Dengue shock is unlikely to occur once renal function improving
- Platelets above 20 * 10^9/l are considered safe for discharge
- Stable haematocrit is essential for discharge

Submit answer

Reference ranges

Score: **22%**

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Which of the following is true regarding his discharge?

- Dengue shock is unlikely to occur after 24 hours post-fever — 8%
- Dengue shock is unlikely to occur once platelets improving — 30%
- Dengue shock is unlikely to occur once renal function improving — 12%
- Platelets above 20 * 10^9/l are considered safe for discharge — 6%
- **Stable haematocrit is essential for discharge** — 44%

This gentleman is admitted in the febrile phase of his dengue infection. The plasma leak phase may occur anytime within the first 48 hours after the fever has broken and manifests with leakage into pleural and peritoneal spaces. It may be accompanied by shock and in some cases haemorrhage. Platelets and renal function may be falsely reassuring as they tend to improve after

the febrile phase but rising haematocrit is a sensitive sign of plasma leak. It is therefore essential to monitor patients for a full 48 hours after the febrile phase and ensure haematocrit is stable prior to discharge. Platelets should be at least 50 * 10^9/l and rising prior to discharge.

Reference: WHO guidelines for clinical management of Dengue infection

Discuss (4) | Improve

Next question >

Dengue fever ★

Dengue fever is a viral infection that can progress to viral haemorrhagic fever (other examples include yellow fever, Lassa fever, Ebola).

Aetiology

- dengue virus is a RNA virus of the genus Flavivirus
- transmitted by the *Aedes aegypti* mosquito
- incubation period of 7 days

Clinical features

Patients with dengue fever can be classified as follows:

- dengue fever:
  - without warning signs
  - with warning signs
- severe dengue (dengue haemorrhagic fever)

Dengue fever

- fever
- headache (often retro-orbital)
- myalgia, bone pain and arthralgia ('break-bone fever')
- pleuritic pain
- facial flushing (dengue)
- maculopapular rash
- haemorrhagic manifestations e.g. positive tourniquet test, petechiae, purpura/ecchymosis, epistaxis
- 'warning signs' include:
  - abdominal pain
  - hepatomegaly
  - persistent vomiting
  - clinical fluid accumulation (ascites, pleural effusion)

Severe dengue (dengue haemorrhagic fever)

- this is a form of disseminated intravascular coagulation (DIC) resulting in:
  - thrombocytopenia
  - spontaneous bleeding
- around 20-30% of these patients go on to develop dengue shock syndrome (DSS)

Investigations

- typically blood results
  - leukopenia, thrombocytopenia, raised aminotransferases
- diagnostic tests
  - serology
  - nucleic acid amplification tests for viral RNA
  - NS1 antigen test

Treatment

- entirely symptomatic e.g. fluid resuscitation, blood transfusion etc
- no antivirals are currently available



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians

8 14

2011 Dengue fever review

Postgraduate Medical Journal

Review of dengue fever

6 9

Suggest link Report broken link

Score: **16.8%**

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A 28-year-old woman presents to the dermatology clinic with a rash on her right upper arm. She says it has been enlarging over the past 2 weeks. There is no history of pain or itch. Her past medical history includes mild gastritis and she is 14-weeks pregnant. She has no known allergies.

On examination, her observations are within normal limits. Her rash is examined, as shown below:



What is the recommended management for this patient?

- Amoxicillin
- Clotrimazole
- Doxycycline
- Phenoxymethylpenicillin
- Prednisolone

Submit answer

Reference ranges

Score: **22%**

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On examination, her observations are within normal limits. Her rash is examined, as shown below:



What is the recommended management for this patient?

Amoxicillin	39%
Clotrimazole	2%
Doxycycline	51%
Phenoxymethylpenicillin	5%
Prednisolone	3%

This patient's rash is typical of erythema migrans given the 'bulls-eye' appearance of a circular

erythematous centre with a clear border and outer erythematous border. Erythema migrans is typically associated with Lyme disease, a zoonotic infection secondary to the spirochete *Borellia burgdorferi*. It is spread by the bites of infected ticks. Associated symptoms include fever, myalgia and fatigue. The rash can appear 1-2 weeks following a tick bite and can enlarge in size over time with central clearing. The lesions are not typically painful or itchy.

Amoxicillin is correct. The typical first-line treatment for Lyme disease is doxycycline. However, as this patient is pregnant, doxycycline is contraindicated and amoxicillin is the next best treatment of choice.

Clotrimazole is incorrect. This is used in the treatment of fungal infections including tinea corporis. Tinea corporis can present similarly with a circular lesion and an erythematous outer border. However, the lesions with ringworm are usually itchy with a scaly outer edge which is not seen here.

Doxycycline is incorrect. This is the first-line management for lyme disease. However, doxycycline is contraindicated in pregnancy due to the risk of bone and teeth deformity. Therefore, the most appropriate option would be amoxicillin.

Phenoxymethylpenicillin is incorrect. This is typically used in the management of infections caused secondary to Streptococcal species (e.g. *Streptococcus pyogenes*). Group A Streptococci such as *Streptococcus pyogenes* have the potential to cause infections such as tonsillitis and rheumatic fever. Rheumatic fever is associated with erythema marginatum, an annular erythematous rash that appears on the trunk, upper arms and legs that spread outwards in a circular shape with central clearing. However, this is very rare in adults and in developed countries.

Prednisolone is incorrect. Corticosteroids can be used for several inflammatory skin conditions including eczema and psoriasis. However, these lesions are often multiple, exfoliative, scaly and itchy which is not seen in this patient's presentation. Therefore, an alternative diagnosis should be considered.

Discuss (2) | Improve

Next question >

Lyme disease ★

Lyme disease is caused by the spirochaete *Borrelia burgdorferi* and is spread by ticks.

Early features (within 30 days)

- erythema migrans
  - 'bulls-eye' rash is typically at the site of the tick bite
  - typically develops 1-4 weeks after the initial bite but may present sooner
  - usually painless, more than 5 cm in diameter and slowlly increases in size

    - present in around 80% of patients.
- systemic features
    - headache
    - lethargy
    - fever
    - arthralgia





Later features (after 30 days)

- cardiovascular
    - heart block
    - peri/myocarditis
- neurological
    - facial nerve palsy
    - radicular pain
    - meningitis



Investigation

- NICE recommend that Lyme disease can be diagnosed clinically if erythema migrans is present
    - erythema migrans is therefore an indication to start antibiotics
- enzyme-linked immunosorbent assay (ELISA) antibodies to *Borrelia burgdorferi* are the first-line test
    - if negative and Lyme disease is still suspected in people tested within 4 weeks from symptom onset, repeat the ELISA 4-6 weeks after the first ELISA test. If still suspected in people who have had symptoms for 12 weeks or more then an immunoblot test should be done
    - if positive or equivocal then an immunoblot test for Lyme disease should be done

Management of asymptomatic tick bites

- tick bites can be a relatively common presentation to GP practices, and can cause significant anxiety
- if the tick is still present, the best way to remove it is using fine-tipped tweezers, grasping the tick as close to the skin as possible and pulling upwards firmly. The area should be washed

following.

- NICE guidance does not recommend routine antibiotic treatment to patients who've suffered a tick bite

Management of suspected/confirmed Lyme disease

- doxycycline if early disease. Amoxicillin is an alternative if doxycycline is contraindicated (e.g. pregnancy)
  - people with erythema migrans should be commenced on antibiotic without the need for further tests
- ceftriaxone if disseminated disease
- Jarisch-Herxheimer reaction is sometimes seen after initiating therapy: fever, rash, tachycardia after first dose of antibiotic (more commonly seen in syphilis, another spirochaetal disease)



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 11 13

Lyme disease: management

NICE 11 11

2018 Lyme disease guidelines

Dermnet NZ 12 5

Lyme disease

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Media



Lyme disease

Osmosis - YouTube 4 1

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Score: **16.8%**

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A 45-year-old female presents with a rash. She states that she initially felt feverish and had a runny nose. This progressed to a rash over her arms, trunk and legs. She recently immigrated to the UK from Poland. She lived in a travellers community in Poland and is not aware of any childhood vaccinations. She has a past medical history of asthma and hereditary spherocytosis. On examination you note a erythematous maculopapular rash over the the upper limbs, thorax and lower limbs.

The following investigations were performed:

Hb	95 g/l	Na^+	138 mmol/l
Platelets	110 * 10^9/l	K^+	3.8 mmol/l
WBC	3.8 * 10^9/l	Urea	6.5 mmol/l
Neuts	1.8 * 10^9/l	Creatinine	58 µmol/l
Lymphs	1.6 * 10^9/l	CRP	48 mg/l

What is the most likely diagnosis?

- Measles
- Mumps
- Rubella
- Parvovirus B19
- Scabies

Submit answer

Reference ranges

Score: **22%**

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Lymphs	1.6 * 10^9/l	CRP	48 mg/l

What is the most likely diagnosis?

Measles	47%
Mumps	2%
Rubella	10%
Parvovirus B19	40%
Scabies	0%

Parvovirus B19 can cause fever, rash and patients with predisposing haematological conditions, pancytopenia

Important for me Less important

The patient has presented with a febrile rash. The differential is wide. The immunisation history should alert you to possible measles or rubella. However the presence of pancytopenia favours a diagnosis of parvovirus B19 infection which is associated with an aplastic crisis.

Although most patients have a decrease of hematopoiesis during parvovirus infection, it is most dangerous in patients with pre-existing bone marrow stress (e.g. sickle cell anaemia or hereditary

spherocytosis) as these disorders are heavily dependent on erythropoiesis due to the reduced lifespan of the red cells.

Discuss (8) | Improve

Next question >

Parvovirus B19

Parvovirus B19 is a DNA virus which causes a variety of clinical presentations. It was identified in the 1980's as the cause of erythema infectiosum

Erythema infectiosum (also known as fifth disease or 'slapped-cheek syndrome')

The illness may consist of a mild feverish illness which is hardly noticeable. However, in others there is a noticeable rash which appears after a few days. The rose-red rash makes the cheeks appear bright red, hence the name 'slapped cheek syndrome'. The rash may spread to the rest of the body but unlike many other rashes, it only rarely involves the palms and soles.

The child begins to feel better as the rash appears and the rash usually peaks after a week and then fades. The rash is unusual in that for some months afterwards, a warm bath, sunlight, heat or fever will trigger a recurrence of the bright red cheeks and the rash itself. Most children recover and need no specific treatment. School exclusion is unnecessary as the child is not infectious once the rash emerges. In adults, the virus may cause acute arthritis.

Be aware that the virus can affect an unborn baby in the first 20 weeks of pregnancy. If a woman is exposed early in pregnancy (before 20 weeks) she should seek prompt advice from whoever is giving her antenatal care as maternal IgM and IgG will need to be checked. It is spread by the respiratory route and a person is infectious 3 to 5 days before the appearance of the rash. Children are no longer infectious once the rash appears and there is no specific treatment.

The child need not be excluded from school as they are no longer infectious by the time the rash occurs.

Other presentations

Other presentations include:

- asymptomatic
- pancytopaenia in immunosuppressed patients
- aplastic crises e.g. in sickle-cell disease
    - parvovirus B19 suppresses erythropoiesis for about a week so aplastic anaemia is rare unless there is a chronic haemolytic anaemia

- hydrops fetalis
  - parvovirus B19 in pregnant women can cross the placenta in pregnant women
  - this causes severe anaemia due to viral suppression of fetal erythropoiesis → heart failure secondary to severe anaemia → the accumulation of fluid in fetal serous cavities (e.g. ascites, pleural and pericardial effusions)
  - treated with intrauterine blood transfusions

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ 9 4

Fifth disease

Suggest link Report broken link

Media



Parvovirus B19

Osmosis - YouTube 4 0

Report broken media

Score: **16.8%**

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Back to top

A 42-year-old man presents with a fever and shortness of breath. Of note, he is a cattle farmer. Observations are as follows: heart rate 82 beats per minute, blood pressure 130/85 mmHg, temperature 38.2ºC, respiratory rate 20 breaths per minute, and SpO2 95% on air.

Blood results are as follows:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	162 * 10^9/L	(150 - 400)
WBC	14.4 * 10^9/L	(4.0 - 11.0)
Na^+	136 mmol/L	(135 - 145)
K^+	3.8 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)
CRP	44 mg/L	(< 5)

Bilirubin	16 µmol/L	(3 - 17)
ALP	104 u/L	(30 - 100)
ALT	146 u/L	(3 - 40)
Î³GT	68 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

A chest x-ray is as follows:

Chest x-ray	Perihilar patchy reticulonodular opacities

What treatment is indicated?

- Amoxicillin
- Clarithromycin
- Co-amoxiclav
- Doxycycline

Levofloxacin

Submit answer

Reference ranges

Score: **22%**

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A chest x-ray is as follows:

Chest x-ray	Perihilar patchy reticulonodular opacities

What treatment is indicated?

- Amoxicillin 2%
- Clarithromycin 14%
- Co-amoxiclav 3%
- Doxycycline 74%

Levofloxacin 6%

Doxycycline should be used to treat Q fever

Important for me Less important

Doxycycline is correct. The chest x-ray appearances are suggestive of atypical pneumonia. Atypical pneumonia associated with fever, hepatitis, and exposure to cattle is highly suggestive of Q fever. Doxycycline is the antibiotic of choice for Q fever.

Amoxicillin is incorrect. Penicillins are ineffective against Q fever since the bacteria is intracellular.

Clarithromycin is incorrect. Whilst macrolides have activity against some intracellular bacteria, they are not considered the first-line treatment choice for Q fever due to a limited evidence base.

Co-amoxiclav is incorrect. Penicillins are ineffective against Q fever since the bacteria is intracellular.

Levofloxacin is incorrect. Q fever can be treated with fluoroquinolones however these agents are not first-line since they have a much broader spectrum.

Discuss (4) Improve

Next question

Q fever

Q fever is caused by *Coxiella burnetii*, a rickettsia. The source of infection is typically an abattoir, cattle/sheep or it may be inhaled from infected dust

Features

- typically prodrome: fever, malaise
- causes pyrexia of unknown origin
- transaminitis
- atypical pneumonia
- endocarditis (culture-negative)

Management

- doxycycline

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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Back to top

A 55-year-old man was admitted with a fever, pain, and erythema across his abdomen. He had been discharged 8 days prior following an elective repair of an umbilical hernia. He has a background of type 2 diabetes mellitus and a raised body mass index of 32kg/m^2 (normal range 18.5 - 24.9kg/m^2).

He had been recovering from his operation at home and had felt well up until that morning. He had noticed a small area of erythema and tenderness around the incision site near his umbilicus. This area of erythema seemed to grow and become more painful throughout the day, therefore, he was brought to the Emergency Department by his wife.

On examination, he looked unwell. His observations were as follows:

- temperature 38.6ºC
- respiratory rate 23 breaths/min
- oxygen saturation 97% on air
- heart rate 125bpm
- blood pressure 88/61mmHg

He felt nauseous and had vomited once, complaining of severe abdominal pain. There was a large area of erythema across the lower half of his abdomen which was extremely tender to palpation. Examination of the erythematous area also revealed impaired sensation to light touch.

His blood results:

WBC	17 * 10^9/L	(4.0 - 11.0)
CRP	460 mg/L	(< 5)

What is the most likely cause of this patient's infection?

- Polymicrobial
- *Streptococcus pyogenes*
- *Aeromonas hydrophila*
- *Staphylococcus epidermidis*
- *Bacillus anthracis*

Submit answer

Reference ranges

Score: **22%**

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His blood results:

WBC	17 * 10^9/L	(4.0 - 11.0)
CRP	460 mg/L	(< 5)

What is the most likely cause of this patient's infection?

Polymicrobial	34%
Streptococcus pyogenes	28%
Aeromonas hydrophila	4%
Staphylococcus epidermidis	30%
Bacillus anthracis	4%

Type 1, or polymicrobial, necrotising fasciitis is the most common

Important for me Less important

This patient is systemically unwell with a rapidly progressive soft tissue infection. Diabetes mellitus is a risk factor for necrotising fasciitis and his recent operation served as a cutaneous portal of entry for causative organisms. A rapidly expanding area of cellulitis in a systemically unwell patient should alert you to the possible diagnosis of necrotising fasciitis. Furthermore, the presence of anaesthesia of the area and pain out of keeping with the visible skin changes is also characteristic of this condition. Whilst necrotising fasciitis is often seen in the extremities, it can also occur in the perineum, head, neck, and trunk.

The most common cause of necrotising fasciitis is type 1, caused by polymicrobial infection of subcutaneous tissue (organisms present include *Bacteroides, Peptostreptococcus, E. coli, Enterobacter*, etc). Bacteria are introduced into the skin via minor skin trauma or surgery and track along fascial planes, extending beyond the area of overlying cellulitis.

Type 2 necrotising fasciitis is a monomicrobial infection most commonly caused by *Streptococcus pyogenes*.

Aeromonas hydrophila is a less common cause of necrotising fasciitis, typically associated with freshwater exposure.

Staphylococcus epidermidis is a commensal organism present on the skin and the most common cause of infections from indwelling medical devices.

Bacillus anthracis, a gram-positive bacteria, causes cutaneous anthrax. There is typically a history of exposure to animals/animal products or IV drug use. A papule forms following exposure which develops into a black eschar with surrounding oedema.

Discuss (3) Improve

Next question ›

Necrotising fasciitis ★

Necrotising fasciitis is a medical emergency that is difficult to recognise in the early stages.

It can be classified according to the causative organism:

- type 1 is caused by mixed anaerobes and aerobes (often occurs post-surgery in diabetics). This is the most common type
- type 2 is caused by *Streptococcus pyogenes*

Risk factors

- skin factors: recent trauma, burns or soft tissue infections
- diabetes mellitus
  - the most common preexisting medical condition
  - particularly if the patient is treated with SGLT-2 inhibitors
- intravenous drug use
- immunosuppression

The most commonly affected site is the perineum (Fournier's gangrene).

Features

- acute onset
- pain, swelling, erythema at the affected site
  - often presents as rapidly worsening cellulitis with pain out of keeping with physical features
  - extremely tender over infected tissue with hypoaesthesia to light touch
  - skin necrosis and crepitus/gas gangrene are late signs
- fever and tachycardia may be absent or occur late in the presentation

Management

- urgent surgical referral debridement
- intravenous antibiotics

Prognosis

- average mortality of 20%

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

5 6

Necrotising fasciitis

Suggest link

Report broken link

Score: **16.8%**

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Back to top

A 48-year-old man presented to the emergency department with a headache, confusion, and slurred speech. His past medical history, family history, and medication history were unremarkable. He had returned to the UK three weeks ago from Germany where he had been camping in a forest with his family. On the last day of his trip, he noticed a tick on his right leg which he carefully removed with tweezers. One week later, he began to develop fevers, muscle aches, and fatigue so was prescribed doxycycline by his GP. After about 7 days these symptoms resolved but over the past 24 hours, he began to feel worse. His family is in good health and he denied any unwell contacts.

On examination, he was confused with a score of 14/15 on the Glasgow Coma Scale and a noticeable tremor. He was globally weak and looked lethargic. His observations were as follows:

- temperature 38.5ºC
- respiratory rate 19/min
- heart rate 92bpm
- blood pressure 128/84mmHg
- oxygen saturations 98% on room air

Examination of his chest and abdomen was unremarkable and no rashes could be seen.

A CT head scan was unremarkable.

A lumbar puncture was performed and the patient was started on empirical treatment with IV aciclovir and ceftriaxone. The CSF results were as follows:

Polynuclear WBCs	3 cells/µL	(0-5 cells/µL)
Mononuclear WBCs	71 cells/µL	(0-5 cells/µL)
Red blood cells	8 cells/µL	(0-10 cells)
Glucose	4.5 mmol/L	(>70% serum glucose)
Protein	0.89 g/L	(0.15-0.45 g/L)
Cytomegalovirus CSF PCR	negative	
Enterovirus CSF PCR	negative	
Epstein-barr virus CSF PCR	negative	
Herpes simplex virus 1 and 2 CSF PCR	negative	
Varicella zoster virus CSF PCR	negative	

Blood test results were as follows:

Serum glucose	5.1 mmol/L	(4 - 11.1 mmol/L)

Borrelia burgdorferi IgG	negative	
Borrelia burgdorferi IgM	negative	
Treponema pallidum screening serology	negative	
Tuberculosis IGRA	negative	
HIV screening serology	negative	

What is the most appropriate management of this patient given the likely diagnosis?

- ○ A further course of doxycycline ✕
- ○ Continue aciclovir ✕
- ○ Supportive management ✕
- ○ IV immunoglobulin ✕
- ○ Praziquantel ✕

Submit answer

Reference ranges

Score: **22%**

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What is the most appropriate management of this patient given the likely diagnosis?

A further course of doxycycline	19%
Continue aciclovir	13%
Supportive management	50%
IV immunoglobulin	10%
Praziquantel	9%

Treatment for tick-borne encephalitis is supportive

Important for me Less important

This patient presents with a history typical of tick-borne encephalitis. Tick-borne encephalitis (TBE) presents as meningoencephalitis, caused by a flavivirus infection of the central nervous system. This virus is transmitted by the *Ixodes* tick, the activity of which is usually highest in the summer months. The disease is endemic in Central and Eastern Europe and the Far East (Russia, China, northern Japan). The disease starts 4-28 days after being bitten by the infected tick and presents in a biphasic fashion. After a few days of fever, fatigue, and myalgia, the patient will have a symptom-free interval before progressing to central nervous system dysfunction (meningitis/encephalitis/cranial nerve palsies). Diagnosis is generally by CSF/serum serology for TBE-specific antibody and management is supportive - aciclovir is given initially as an empirical treatment but can be stopped when HSV/VZV is ruled out.

In this case, whilst the specific serology confirming the infection is not given, the other investigations and history of a tick bite point towards this diagnosis. He has a predominantly lymphocytic pleocytosis with a raised CSF protein which is typical for viral CNS infections.

Whilst doxycycline appeared to help this patient initially, this coincided with the symptom-free interval commonly seen in the biphasic illness of TBE. Negative serology for *Borrelia burgdorferi* makes a diagnosis of Lyme disease very unlikely. Therefore, another course of doxycycline is not appropriate.

Continuing aciclovir is not necessary given that the CSF investigations for HSV-1 and HSV-2 and

VZV are negative.

IV immunoglobulin can be used for autoimmune/paraneoplastic encephalitis but there is nothing in the stem to suggest that this is the underlying pathological process.

Praziquantel is a drug used in the treatment of parasitic infections such as schistosomiasis.

The management of TBE is supportive. Whilst it would be important to identify the cause by performing further serological testing, this is not an option.

Discuss (3) | Improve

Next question >

Tick-borne encephalitis ★

Carried by ticks hosted by native wildlife, tick-borne encephalitis is actually caused by a virus (Flavivirus) and transmitted by the bite of an infected tick.

Once transmitted to the host, flavivirus infection manifests as a biphasic illness. During the first phase, patients may describe constitutional upset reporting headaches, myalgia, and fevers. There is typically then an asymptomatic period before the disease course progresses to phase two, which is characterized by infection by symptoms of central nervous system involvement, typically meningitis or, as the name suggests, encephalitis. The time period between phase one and two may be highly variable or absent in the case of severe infection.

Symptoms usually manifest within 7 days of infection, however, the incubation period can be up to a month. Long term neurological sequelae may persist for months - years following infection.

There are three species of flavivirus implicated in tick-borne encephalitis: European, Far Eastern and Siberian, with the Far Eastern species typically causing the most severe illness, often progressing rapidly to central nervous system involvement with no asymptomatic period, (European centre for Disease Prevention and Control quotes a mortality rate of around 35% in cases of Far Eastern flavivirus species infection).

Diagnosis is made on the basis of clinical suspicion, with patients showing characteristic symptoms and travel history confirming recent travel to affected areas. Diagnosis may be confirmed by cerebrospinal fluid (CSF) analysis demonstrating specific IgM or IgG antibodies.

Treatment is supportive, however, the addition of doxycycline or a cephalosporin may be advised if Lyme disease is considered a differential diagnosis until confirmation via CSF sampling can be obtained.

A vaccination available and recommended to those travelling to endemic areas and planning to

engage in high-risk outdoor activities, for example, hiking in rural forested areas and/or grasslands. Precautions to avoid tick bites are recommended to all travellers to endemic areas.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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A 55-year-old woman is referred for an urgent assessment by the infectious diseases team after presenting to her local out of hours GP. The patient stated that she was concerned about having caught Lyme disease during a recent day out in the New Forest in Hampshire, UK. The patient was particularly concerned about this diagnosis as she had a close friend who had been forced to give up work after suffering from neuroborreliosis.

On closer questioning, the patient reported that 2 days previously she had gone on a walk in the New Forest. When she had arrived home that evening she had found two engorged ticks attached to her right lower leg. Her husband had removed the ticks by applying nail polish to them and then pulling them off with tweezers.

The patient denied suffering from any symptoms at the time of presentation though admitted she was very anxious. In particular, the patient had not experienced any neurological or cardiovascular symptoms and had not observed any skin rashes.

The patient was receiving treatment from her GP for hypercholesterolaemia and mild asthma. The patient had no known drug allergies and was employed as a legal secretary at a local firm.

A full examination of the patient's skin was performed and no abnormal rashes or other skin changes were identified. No focal neurology was identified on examination of the peripheral and central nervous systems. The assessment did not identify any signs of meningism. The patient was afebrile.

What is the appropriate management of this patient's tick bites?

- Treat with amoxicillin for 14 days
- Immediate serological testing for pathogenic *Borrelia* species
- Serological testing for pathogenic *Borrelia* species in 4 weeks (to allow for seroconversion)
- No investigation or treatment indicated at present time, carefully observe for onset of symptoms
- Treat with doxycycline for 14 days

Submit answer

Reference ranges

Score: **22%**

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What is the appropriate management of this patient's tick bites?

Treat with amoxicillin for 14 days	1%
Immediate serological testing for pathogenic *Borrelia* species	9%
Serological testing for pathogenic *Borrelia* species in 4 weeks (to allow for seroconversion)	12%
No investigation or treatment indicated at present time, carefully observe for onset of symptoms	58%
Treat with doxycycline for 14 days	19%

There is no need for prophylactic antibiotics for Lyme disease in asymptomatic patients bitten by a tick

This patient has a convincing history for the attachment of ticks from a region of the UK at high risk of Lyme borreliosis. When assessing the risk of tick bites it is important to clarify for how long the tick was attached and if it became engorged. Transmission of pathogenic *Borrelia* species is unlikely if ticks are attached for less than 24 hours or do not become engorged. Ticks should be removed gently using tweezers to minimise irritation to the skin and the risk of rupturing the tick which can increase the risk of infection.

Asymptomatic patients who report a tick bite should not undergo serological testing or receive treatment. Testing can lead to false positives secondary to previous infection or cross-reactive antibodies. Prophylactic treatment of tick bites may be required if the patient is immunosuppressed or in certain regions of the USA where the prevalence of infected ticks is particularly high.

When serological testing is indicated paired blood samples should be taken at four week intervals to allow seroconversion to take place. Treatment with doxycycline or amoxicillin for 14 days is the appropriate treatment for a patient presenting with erythema migrans after a tick bite.

Duncan C, Carle G, Seaton R. Tick bite and early Lyme borreliosis. *BMJ* 2012;344:e3124.

Discuss (2) Improve

Next question >

Lyme disease

Lyme disease is caused by the spirochaete *Borrelia burgdorferi* and is spread by ticks.

Early features (within 30 days)

- erythema migrans
  - 'bulls-eye' rash is typically at the site of the tick bite
  - typically develops 1-4 weeks after the initial bite but may present sooner
  - usually painless, more than 5 cm in diameter and slowlly increases in size
  - present in around 80% of patients.
- systemic features
  - headache
  - lethargy
  - fever
  - arthralgia





Later features (after 30 days)

- cardiovascular
  - heart block
  - peri/myocarditis
- neurological
  - facial nerve palsy
  - radicular pain
  - meningitis



Investigation

- NICE recommend that Lyme disease can be diagnosed clinically if erythema migrans is present
  - erythema migrans is therefore an indication to start antibiotics
- enzyme-linked immunosorbent assay (ELISA) antibodies to *Borrelia burgdorferi* are the first-line test
  - if negative and Lyme disease is still suspected in people tested within 4 weeks from symptom onset, repeat the ELISA 4-6 weeks after the first ELISA test. If still suspected in people who have had symptoms for 12 weeks or more then an immunoblot test should be done
  - if positive or equivocal then an immunoblot test for Lyme disease should be done

Management of asymptomatic tick bites

- tick bites can be a relatively common presentation to GP practices, and can cause significant anxiety
- if the tick is still present, the best way to remove it is using fine-tipped tweezers, grasping the tick as close to the skin as possible and pulling upwards firmly. The area should be washed following.
- NICE guidance does not recommend routine antibiotic treatment to patients who've suffered a tick bite

Management of suspected/confirmed Lyme disease

- doxycycline if early disease. Amoxicillin is an alternative if doxycycline is contraindicated (e.g. pregnancy)

    - people with erythema migrans should be commenced on antibiotic without the need for further tests
- ceftriaxone if disseminated disease
- Jarisch-Herxheimer reaction is sometimes seen after initiating therapy: fever, rash, tachycardia after first dose of antibiotic (more commonly seen in syphilis, another spirochaetal disease)



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries — 11 13

Lyme disease: management

NICE — 11 11

2018 Lyme disease guidelines

Dermnet NZ — 12 5

Lyme disease

Suggest link | Report broken link

Media



Lyme disease

Osmosis - YouTube 4 1

Report broken media

Score: **16.8%**

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A 14-year-old, unvaccinated child is brought into the emergency department after possible exposure to measles. The child has juvenile arthritis well controlled on methotrexate but is otherwise well with no other medical issues. His mother explains that the child attended a large party 3 days previously and she received a call this morning informing them that another child who attended the party has just been diagnosed with measles. She has no further information regarding the other child or as to whether they were symptomatic etc.

The 14-year-old reports no symptoms currently and appears well in the emergency department. His examination is unremarkable and observations are as follows:

- Heart rate 100 beats per minute
- Blood pressure 120/80 mmHg
- Respiratory rate 20 breaths per minute
- Oxygen saturations 98%
- Temperature 36.2 ºC

What is the most appropriate management option?

- No treatment is indicated
- Provide aciclovir immediately
- Provide immunoglobulin immediately
- Test for IgM antibodies and if present start aciclovir
- Test for serum measles DNA and if present provide immunoglobulin

Submit answer

Reference ranges

Score: **22%**

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- Heart rate 100 beats per minute
- Blood pressure 120/80 mmHg
- Respiratory rate 20 breaths per minute
- Oxygen saturations 98%
- Temperature 36.2 ºC

What is the most appropriate management option?

No treatment is indicated	18%
Provide aciclovir immediately	4%
Provide immunoglobulin immediately	54%
Test for IgM antibodies and if present start aciclovir	12%
Test for serum measles DNA and if present provide immunoglobulin	13%

Those exposed to measles can be given immunoglobulin to reduce the risk of infection

Important for me Less important

Providing immunoglobulin (Ig) is correct as the child is unvaccinated, immunocompromised due to methotrexate and has had exposure to measles. The MMR vaccination can also be offered to patients who are unvaccinated and have exposure as vaccine-induced measles antibody develops more rapidly than that following natural infection.

No treatment is incorrect. The child is unvaccinated, immunocompromised and most likely had significant measles exposure therefore immunoglobulin should be provided as a minimum.

Aciclovir is incorrect as it does not play a role in post-exposure prophylaxis or the treatment of active measles.

Testing for serum measles DNA is incorrect as measles is an RNA paramyxovirus and also this will have no impact on management.

IgM antibodies can be tested for, and are normally present within a few days of rash onset however aciclovir is not indicated in measle post-exposure prophylaxis.

Discuss (4) Improve

Next question

Measles

Measles is now rarely seen in the developed world following the adoption of immunisation programmes. Outbreaks are occasionally seen, particularly when vaccinations rates drop, for example after the MMR controversy of the early 2000s.

Overview

- RNA paramyxovirus
- one of the most infectious known viruses
- spread by aerosol transmission
- infective from prodrome until 4 days after rash starts
- incubation period = 10-14 days

Features

- prodromal phase
  - irritable
  - conjunctivitis
  - fever
- Koplik spots
  - typically develop before the rash
  - white spots ('grain of salt') on the buccal mucosa
- rash
  - starts behind ears then to the whole body
  - discrete maculopapular rash becoming blotchy & confluent
  - desquamation that typically spares the palms and soles may occur after a week
- diarrhoea occurs in around 10% of patients



© Image used on license from DermNet NZ

Koplik spots

Investigations

- IgM antibodies can be detected within a few days of rash onset

Management

- mainly supportive
- admission may be considered in immunosuppressed or pregnant patients
- notifiable disease → inform public health

Complications

- otitis media: the most common complication
- pneumonia: the most common cause of death
- encephalitis: typically occurs 1-2 weeks following the onset of the illness)
- subacute sclerosing panencephalitis: very rare, may present 5-10 years following the illness
- febrile convulsions
- keratoconjunctivitis, corneal ulceration
- diarrhoea
- increased incidence of appendicitis
- myocarditis



© Image used on license from DermNet NZ

The rash typically starts behind the ears and then spreads to the whole body

Management of contacts

- if a child not immunized against measles comes into contact with measles then MMR should be offered (vaccine-induced measles antibody develops more rapidly than that following natural infection)
- this should be given within 72 hours

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal

5 6

Subacute sclerosing panencephalitis

Suggest link | Report broken link

Media



Measles - What is it?

Let's Learn About Bugs - YouTube 3 0



Measles: Features, Management and Mnemonics

Townsend Teaching - YouTube 2 0



Measles

Osmosis - YouTube 4 1

Report broken media

Score: **16.8%**

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A 30-year-old homeless, intravenous drug user is brought into the emergency department by his concerned friends. The patient has difficulty speaking clearly but his friends report he had been complaining of progressive weakness, especially of his face with difficulty swallowing and some double vision.

On examination, the patient has a broad gait, with flaccid weakness globally. Cranial nerve examination relieves ptosis, ocular and facial muscle weakness. The patient exam is otherwise normal including a normal GCS.

On further questioning, the group who brought the patient in reports another one of their friends was admitted with similar symptoms a few days ago. On review of the hospital computer system, it is noted that this friend's blood cultures grew a gram-positive, anaerobic bacillus.

What should be provided to the patient urgently?

- ○ Intubation and ventilation ×
- ○ Broad spectrum antibiotics ×
- ○ Plasma exchange ×
- ○ Antitoxin ×
- ○ Specific vaccination ×

Submit answer

Reference ranges

Score: **22%**

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What should be provided to the patient urgently?

Intubation and ventilation	9%
Broad spectrum antibiotics	9%
Plasma exchange	7%
Antitoxin	75%
Specific vaccination	1%

The treatment of botulism - supportive care and botulism antitoxin

Important for me Less important

This patient is suffering from botulism which, although rare, normally presents following consumption of contaminated food (e.g. tinned) or as in this case IV drug use. The patient has the typical presentation of flaccid paralysis with bulbar and ocular involvement, whilst maintaining full consciousness and no sensory disturbance. The mainstay of treatment is supportive with early antitoxin use which can neutralise non-bound, circulating botulinum toxin.

This patient has most likely a diagnosis of botulism. Although this can progress to cause respiratory issues the patient does not currently have evidence of respiratory failure. Antitoxin should be given as a priority and respiratory function monitored.

Broad-spectrum antibiotics can be considered if there is evidence of wound infection with *Clostridium botulinum* otherwise there use is non-beneficial and specific botulism antitoxin with supportive care are the only management that will affect the course of the disease.

Plasma exchange is incorrect as this has no evidence for its use in botulism.

To date, no effective vaccination to assist in the management of botulism has been approved for use.

Discuss (3) Improve

Next question ›

Botulism ★

Clostridium botulinum

- gram positive anaerobic bacillus
- 7 serotypes A-G
- produces botulinum toxin, a neurotoxin which irreversibly blocks the release of acetylcholine
- may result from eating contaminated food (e.g. tinned) or intravenous drug use
- neurotoxin often affects bulbar muscles and autonomic nervous system

Features

- patient usually fully conscious with no sensory disturbance
- flaccid paralysis
- diplopia
- ataxia
- bulbar palsy

Treatment

- botulism antitoxin and supportive care
  - antitoxin is only effective if given early - once toxin has bound its actions cannot be reversed

123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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A 23-year-old woman attends with fever, myalgia, rash, and headache 4 days after returning from Sao Paulo. She noted that fevers and generalised abdominal pain started the day before she boarded her flight home and that the rash had come up in the last 24 hours.

On examination, the woman has petechiae and a maculopapular rash over her wrists, ankles, and palms as well as a single itchy, erythematous raised papule with a central punctum on her right calf.

Given the likely diagnosis, what is the most appropriate treatment?

- Albendazole
- Artemether with lumefantrine
- Doxycycline
- Metronidazole
- Praziquantel

Submit answer

Reference ranges

Score: **22%**

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Given the likely diagnosis, what is the most appropriate treatment?

Albendazole	17%
Artemether with lumefantrine	4%
Doxycycline	59%
Metronidazole	2%
Praziquantel	18%

Doxycycline is typically used in the management of typhus

Important for me Less important

The most likely diagnosis here is Rocky Mountain spotted fever, one of the Rickettsial diseases. Rocky Mountain spotted fever is transmitted via tick bites in endemic areas such as South America - the single, itchy, erythematous, raised papule is descriptive of a tick bite. It presents initially with general symptoms of malaise, fever, and abdominal pain. The classic vasculitic rash then develops after 3-5 days.

Doxycycline is the treatment of choice for Rocky Mountain spotted fever.

Albendazole is incorrect. This would be more appropriate for helminth infections such as roundworm and hookworm.

Artemether with lumefantrine is incorrect. This would be the appropriate treatment for uncomplicated *P. falciparum* malaria. *P. falciparum* malaria is most prevalent in Africa rather than in South America. It can present with similar general symptoms to Rocky Mountain spotted fever

with fevers, headache, and myalgias but is also often associated with hepatomegaly and splenomegaly. It is not typically associated with the vasculitic rash seen in this patient.

Metronidazole is incorrect. It has good action against anaerobic bacteria and protozoa but would not be the choice for Rickettsial infections.

Praziquantel is incorrect. This is used to treat parasitic worm infections such as schistosomiasis. It would not be the choice for Rickettsial infections.

Discuss (5) | Improve

Next question >

Typhus

Typhus is a group of rickettsial diseases transmitted between hosts by arthropods. Examples include

- endemic typhus
  - caused by *Rickettsia typhi*
  - reservoir/vector: fleas on rats
  - occurs throughout the world, typically in warm costal regions
  - relative bradycardia
- epidemic typhus
  - caused by *Rickettsia prowazekii*
  - reservoir/vector: body louse
  - more common in central and eastern Africa, central and South America
- scrub typhus
  - caused by *Orientia tsutsugamushi*
  - reservoir/vector: harvest mites on humans or rodents
  - more common in Asia
  - black eschar at site of original inoculation
  - relative bradycardia despite fever
- spotted fever
  - caused by Rickettsia spotted fever group
  - spread by ticks
  - examples include Rocky Mountain spotted fever

Common features

- fever, headache, malaise
- rash
  - typically maculopapular
  - begins on the trunk and spreads to the extremities
- later complications

- meningoencephalitis

Management

- doxycycline

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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A 25-year-old gentleman was referred to the outpatient infectious diseases clinic by his GP. He had recently immigrated from Pakistan a few months ago with his extended family. A couple of weeks ago his mother who lived in the same house as him was diagnosed with respiratory tuberculosis and he was accordingly advised to get 'checked up' by his GP. He felt well in himself, stating that he recovered from a cold a few weeks ago having had symptoms of nasal congestion, sore throat and a cough lasting a few days. He has since felt well, denying the presence of any further symptoms. Of note he has not lost weight, not had a fever, and did not suffer from a cough or night sweats. He was unsure what immunisations he had whilst in Pakistan. As a precautionary measure, the GP referred him to the infectious diseases clinic given the close proximity with his mother.

Examination revealed a well looking 25-year-old gentleman. His temperature was 36.6ºC, heart rate 78 bpm, respiratory rate 16/min and oxygen saturations 99% on air. He had a BCG scar on his left arm. Examination of his cardiovascular, respiratory, gastrointestinal and neurological systems were all entirely unremarkable.

Initial investigations revealed the following:

Blood film:negative; including negative malaria screen

Blood culture: negative culture; awaiting prolonged acid-fast bacilli culture result

Quantaferon blood test: positive
Mantoux test: positive

Chest x-ray: normal appearances of heart and lung fields, no abnormality was seen

Based upon the above information, what is the next best management step?

- Commence treatment with isoniazid, pyridoxine, ethambutol, rifampicin and pyrazinamide for 2 months
- Discharge from clinic with no further action to take
- Refer for diagnostic bronchoscopy alveolar lavage
- Commence isoniazid and pyridoxine for 6 months
- Repeat Quantaferon blood test weekly for the next month - treat if remains positive

Submit answer

Reference ranges

Score: **22%**

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Quantaferon blood test: positive
Mantoux test: positive

Chest x-ray: normal appearances of heart and lung fields, no abnormality was seen

Based upon the above information, what is the next best management step?

Commence treatment with isoniazid, pyridoxine, ethambutol, rifampicin and pyrazinamide for 2 months	14%
Discharge from clinic with no further action to take	8%
Refer for diagnostic bronchoscopy alveolar lavage	6%
Commence isoniazid and pyridoxine for 6 months	66%
Repeat Quantaferon blood test weekly for the next month - treat if remains positive	6%

Latent tuberculosis treatment options:

- 3 months of isoniazid (with pyridoxine) and rifampicin, or
- 6 months of isoniazid (with pyridoxine)

Important for me Less important

This gentleman has latent TB. He has had close (and presumably prolonged) contact with an active TB carrier but other than the presence of transient symptoms of an upper respiratory tract infection has been asymptomatic. He has had a positive Mantoux test, which can be positive with both active and latent TB but can also by a previous BCG vaccination. Quantiferon testing, on the other hand, is not influenced by BCG vaccination status but cannot distinguish between active and latent TB. However in the absence of significant symptoms and with normal investigations including a normal CXR the most likely diagnosis is latent TB and therefore he requires monotherapy with isoniazid (and pyridoxine) for 6 months.

Discuss (1) | Improve

Next question >

Tuberculosis: latent disease

Patients with latent tuberculosis are asymptomatic and non-infectious. They are typically diagnosed by having a positive tuberculin skin test or Interferon-Gamma Release Assay (IGRA) combined with a normal chest x-ray, helping to exclude active tuberculosis.

NICE now give two choices for treating **latent tuberculosis**:

- 3 months of isoniazid (with pyridoxine) and rifampicin, or
- 6 months of isoniazid (with pyridoxine)

Why two choices? NICE (2016) state the following:

> *Base the choice of regimen on the person's clinical circumstances. Offer:*

- *3 months of isoniazid (with pyridoxine) and rifampicin to people younger than 35 years if hepatotoxicity is a concern after an assessment of both liver function (including transaminase levels) and risk factors*
- *6 months of isoniazid (with pyridoxine) if interactions with rifamycins are a concern, for example, in people with HIV or who have had a transplant.*

People with latent tuberculosis cannot pass the disease on to others, so there is no restriction in terms of employment etc. This advice would obviously change if the patient developed active tuberculosis at any point.

Risk factors for developing active tuberculosis include:

- silicosis
- chronic renal failure
- HIV positive
- solid organ transplantation with immunosuppression
- intravenous drug use
- haematological malignancy
- anti-TNF treatment
- previous gastrectomy



Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2016 Tuberculosis guidelines

1 0

Greenbook 0 0

Tuberculosis

Suggest link Report broken link

Media



Tuberculosis

Osmosis - YouTube 2 0

Report broken media

Score: **16.8%**

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A 28-year-old man presents to the Emergency Department with a four-day history of recurrent headaches. He reports no significant medical history but mentions a recent trip to Bolivia, where he recalls sustaining a tick bite. He also describes a rash that started on his extremities.

On examination, you observe a maculopapular rash involving his extremities, face, and trunk. His vital signs are as follows: blood pressure 110/80 mmHg, pulse rate 78 beats per minute, and temperature 39.6 ºC.

The following laboratory results are available:

Hb	140 g/L	Male: (135-180)
Platelets	105 * 10^9/L	(150 - 400)
WBC	3.1 * 10^9/L	(4.0 - 11.0)
ALT	68 u/L	(3 - 40)

What is the most likely diagnosis?

- Babesiosis
- Dengue Fever
- Lyme disease
- Rocky Mountain Spotted Fever
- Tularemia

Submit answer

Reference ranges

Score: **22%**

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Back to top

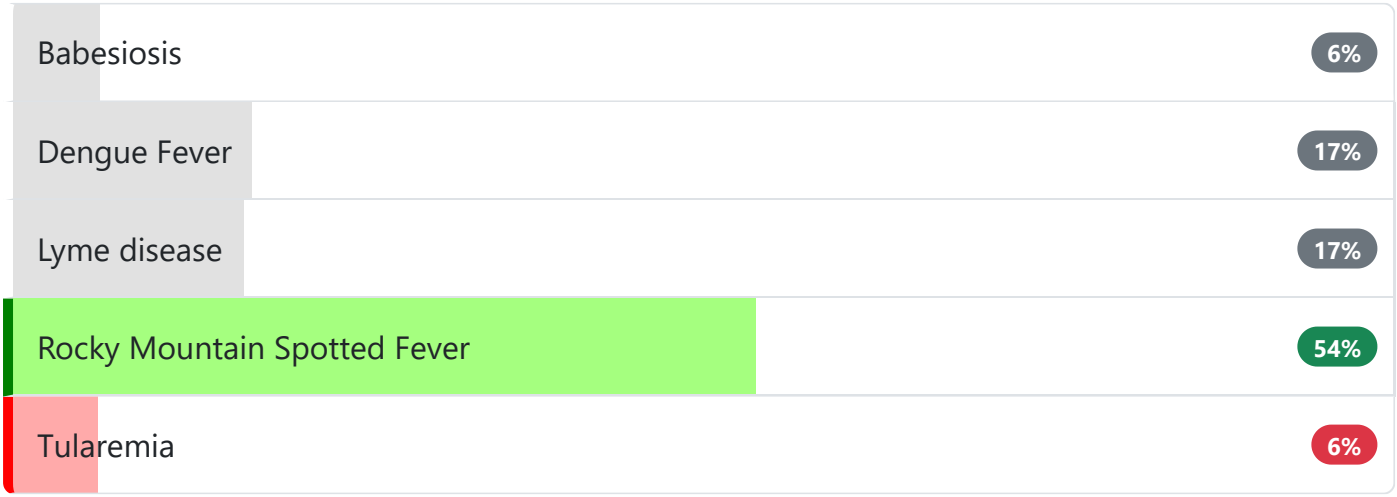
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Platelets	105 * 10^9/L	(150 - 400)
WBC	3.1 * 10^9/L	(4.0 - 11.0)
ALT	68 u/L	(3 - 40)

What is the most likely diagnosis?



Headache, fever, rash on the peripheries before spreading centrally, tick bite → Rocky Mountain spotted fever (*Rickettsia ricketsii*)

Important for me Less important

Rocky Mountain Spotted Fever is the correct diagnosis. The clinical presentation of fever, headache, and a rash that started on the extremities and subsequently spread to the face and trunk, coupled with a travel history to South America (specifically Bolivia) and a tick bite, should prompt consideration of Rocky Mountain spotted fever. This disease involves the proliferation of intracellular bacteria *Rickettsia rickettsii* at the site of the tick bite, predominantly affecting the endothelium of small vessels. Laboratory findings such as thrombocytopenia, leukopenia, and raised liver enzymes further support this diagnosis.

Babesiosis represents a tick-borne disease caused by protozoan parasites of the Babesia genus. Transmission is primarily through Ixode ticks. While it shares common features with Rocky Mountain spotted fever, including fever, headache, thrombocytopenia, and increased liver enzyme levels, Babesiosis characteristically does not present with a rash and mainly infects erythrocytes.

Dengue Fever is an arboviral infection endemic in the Americas transmitted by mosquitoes carrying dengue virus. Clinical manifestations typically include fever, retro-orbital pain, severe myalgia, and a distinctive rash which commences with facial flushing before progressing to a second maculopapular rash on the back of hands and feet extending to the torso. However, this type of rash differs from that described in the scenario provided.

Lyme disease, another tick-borne illness caused by spirochetes of *Borrelia burgdorferi*, is transmitted via hard ticks. It typically presents with an expanding area of localised erythema with central clearing (erythema migrans), often resembling a bull's-eye pattern at the tick bite location. The rash does not match Lyme disease but suggests Rocky Mountain spotted fever.

Tularemia is an uncommon zoonotic infection caused by Francisella tularensis bacteria and transmitted by ticks. It can cause symptoms such as fever, painful ulcerative lesions at inoculation sites with associated regional lymphadenopathy, hepatosplenomegaly, and potentially community-acquired pneumonia. The patient's clinical features do not correspond with those commonly associated with tularemia.

Discuss (1) Improve

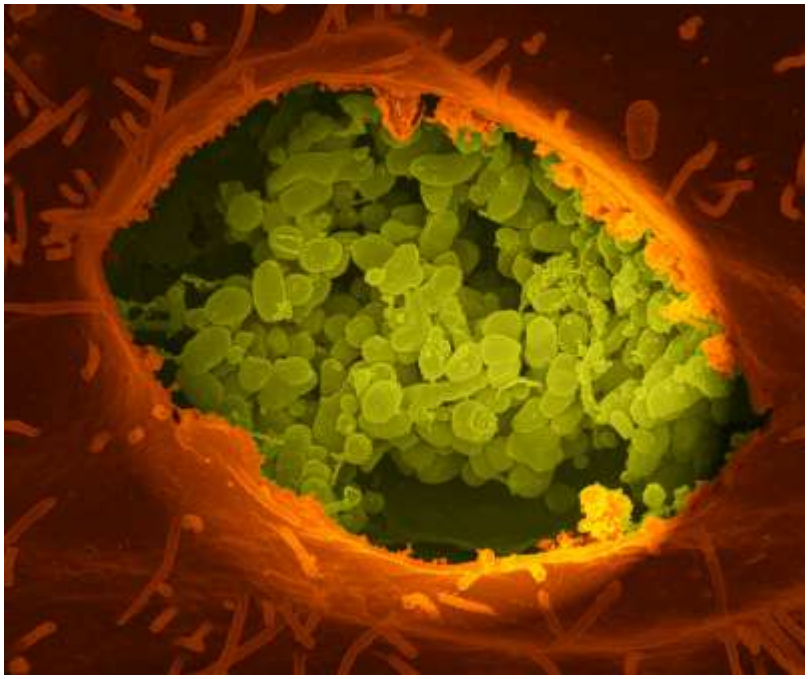
Next question ›

Rickettsiae

Rickettsiae are Gram-negative obligate intracellular parasites. Types of rickettsiae cause a variety of diseases that are typically characterised by fever, headache and rash. A notable exception is Q fever (cause by *Coxiella burnetti* which causes pneumonia but no rash. The Weil-Felix reaction is positive except in Q fever. Rickettsial diseases are all treated with tetracyclines.

Disease	Cause	Vector	Notes
Rocky Mountain spotted fever	*Rickettsia ricketsii*	Tick	Headache and fever are common Rash starts on the peripheries (wrist, ankles) before spreading centrally. It is initially maculopapular before becoming vasculitic Endemic to east coast of US

Disease	Cause	Vector	Notes
Q fever	*Coxiella burnetti*	No vector	No rash but causes pneumonia
Endemic typhus	*Rickettsia typhi*	Flea	Rash starts centrally then spreads to the peripheries
Epidemic typhus	*Rickettsia prowazekii*	Human body louse	
Ehrlichliosis	*Ehrlichia*	Tick	



A dry fracture of a Vero cell exposing the contents of a vacuole where *Coxiella burnetti* are busy growing. Note the intracellular nature of the organism. Credit: NIAID

Next question

Textbooks

High-yield textbook

Extended textbook

Media



Rickettsia rickettsii & other Rickettsia species

Osmosis - YouTube 5 0

Report broken media

Score: **16.8%**

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Back to top

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With the help of three months of nicotine transdermal patches and vareniciline he is now no longer actively smoking. He is married and works as a salesman in a pharmaceutical company. For the last year he has been to many destinations around the world as part of his job.

On examination he appeared ill. He was oriented in time, place, and person, but was mentally slow in understanding commands during examination. The power in the left leg was grade 2 for all muscle groups. Tone, sensation, coordination and reflexes were all normal. The right leg was normal. No abnormality detected in other systems. The following investigation were ordered:

Hb	10g/dl
Platelets	10 * 10^9/l
WBC	4* 10^9/l
Neutrophils	60%
Lymphocytes	34%
Na+	135 mmol/l
K+	4 mmol/l
Creatinine	95 µmol/l
Urea	4 mmol/l

Urine analysis: Clear

MRI brain showed bilateral multiple hyperintense demyelinating lesions involving sub cortical areas without any mass effect.

What is the most appropriate thing to do?

- CSF analysis for oligoclonal bands
- Brain biopsy
- CSF analysis for herpes simplex virus (HSV)
- HIV test

Stop vareniciline

Submit answer

Reference ranges

Score: **22%**

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Urea	4 mmol/l

Urine analysis: Clear

MRI brain showed bilateral multiple hyperintense demyelinating lesions involving sub cortical areas without any mass effect.

What is the most appropriate thing to do?

CSF analysis for oligoclonal bands	15%
Brain biopsy	3%
CSF analysis for herpes simplex virus (HSV)	11%
HIV test	64%

Stop vareniciline 8%

HIV, neuro symptoms, widespread demyelination - progressive multifocal leukoencephalopathy

Important for me Less important

The progressive neurological symptoms with demyelination on MRI points to progressive multifocal leukoencephalopathy (PML).

PML is a demyelinating disease of the CNS due to the JC virus. It occurs almost exclusively in immunosuppressed individuals with the majority of cases being secondary to HIV infection. Patients typically experience insidious onset of focal symptoms that include mainly behavior, cognitive, speech, and motor functions. Polymerase chain reaction (PCR) of the CSF has a high sensitivity and specificity for the detection of JC virus in patients with PML.

Neither the symptoms nor the signs are compatible with either multiple sclerosis or HSV.

Varenicline could cause low platelets and appetite changes with psychiatric manifestation. But it would not explain the motor deterioration nor the cognitive decline for this patient.

Discuss (8) Improve

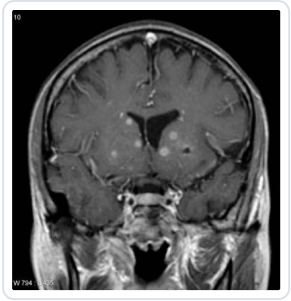
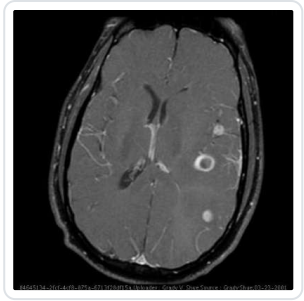
Next question

HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

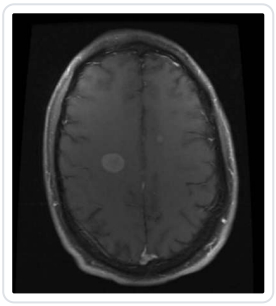
- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination
- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link Report broken link

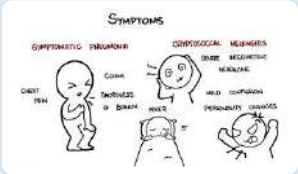
Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

0 1

Report broken media

Score: **16.8%**

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Back to top

A 21-year-old man presents to the emergency department with five days of feeling tired, cough, fevers and general lethargy. He becomes quite tearful during the consultation as he has never felt like this.

On examination, he has tenderness over both trapezium muscles, his temperature is 38.6 ºC and his throat is red without any collections of pus.

A rapid point of care test shows that he is influenza positive.

What is your next management step?

- ○ Oseltamivir ×
- ○ Zanamivir ×
- ○ Admit for observation ×
- ○ Discharge with reassurance ×
- ○ Oral amoxicillin ×

Submit answer

Reference ranges

Score: **22%**

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A rapid point of care test shows that he is influenza positive.

What is your next management step?

Oseltamivir	46%
Zanamivir	3%
Admit for observation	5%
Discharge with reassurance	45%
Oral amoxicillin	1%

Low-risk patients with influenza who present 48 hours after symptoms are recommended for supportive treatment

Important for me Less important

This is a simple case of influenza of which most people who are low risk will recover with supportive treatment. In high risk groups e.g. immunosuppression or pregnancy, oseltamivir or zanamivir can be considered. The duration of symptoms is important as those who present within 48 hours of symptoms are more likely to derive benefit. Oral antibiotics are not usually recommended though bacterial superinfection can occur in influenza.

Discuss (8) Improve

Next question

Influenza

Features

The following are typically seen:

- fever greater than 38ºC
- myalgia
- lethargy
- headache
- rhinitis
- sore throat
- cough
- diarrhoea and vomiting

Management

Consider prescribing antiviral treatment for influenza if all of the following apply:

- 1) The patient is in an at-risk group or is felt to be at risk of developing a serious complication
  - > 65 years old
  - pregnant women
  - chronic disease of respiratory, cardiac, renal, hepatic or neurological nature
  - diabetes
  - immunosuppression
  - morbid obesity.
- 2) There is circulating influenza nationally
  - this would mean typically the winter months
- 3) The patient is able to start treatment within 48 hours from the onset of symptoms

Antivirals for influenza (simplified, please see the CKS link for more details)

- First line: oseltamivir
- Second line: zanamivir



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 0 0

Scenario: Treatment of influenza

Suggest link Report broken link

Media



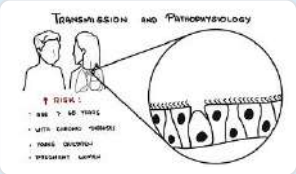
Influenza

Osmosis - YouTube 4 0



Flu - What is it?

Let's Learn About Bugs - YouTube 3 0



Understanding Influenza - The Flu

Armando Hasudungan - YouTube 0 0

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A 27 year-old Lithuanian male presents to the Emergency Department with a short history productive cough, fever and breathlessness. The chest X-Ray shows left upper zone consolidation. His white cell count and CRP are elevated and IV antibiotics are commenced for a community acquired pneumonia. He makes some improvement after 48 hours but remains on 4L of oxygen to maintain adequate saturations. An interferon gamma release assay for mycobacterium is positive.

What would be the correct course of action?

- Send three morning sputum samples for acid fast bacilli
- Arrange a bronchoscopy and washings for acid fast bacilli
- No action, continue antibiotics, repeat CXR in 6 weeks time
- Treat for latent pulmonary tuberculosis with two agents
- Treat for active pulmonary tuberculosis with quadruple therapy

Submit answer

Reference ranges

Score: **22%**

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What would be the correct course of action?

Send three morning sputum samples for acid fast bacilli	46%
Arrange a bronchoscopy and washings for acid fast bacilli	11%
No action, continue antibiotics, repeat CXR in 6 weeks time	3%
Treat for latent pulmonary tuberculosis with two agents	11%
Treat for active pulmonary tuberculosis with quadruple therapy	29%

This young gentleman's positive interferon release assay shows he has been exposed to tuberculosis in the past. It however does not give any information regarding this active infection which could represent pulmonary tuberculosis or bacterial pneumonia. A wise course of action would include isolating the patient and sending three morning sputum samples to look for acid fast bacilli. He is not currently fit for bronchoscopy given his oxygen requirements.

Discuss (9) | Improve

Next question >

Tuberculosis: screening and diagnosis

Screening for latent tuberculosis

The Mantoux test is the main technique used to screen for latent tuberculosis. In recent years the interferon-gamma blood test has also been introduced. It is used in a number of specific situations such as:

- the Mantoux test is positive or equivocal
- people where a tuberculin test may be falsely negative (see below)

Mantoux test

- 0.1 ml of 1:1,000 purified protein derivative (PPD) injected intradermally
- result read 2-3 days later

Diameter of induration	Positivity	Interpretation
< 6mm	Negative - no significant hypersensitivity to tuberculin protein	Previously unvaccinated individuals may be given the BCG
6 - 15mm	Positive - hypersensitive to tuberculin protein	Should not be given BCG. May be due to previous TB infection or BCG
> 15mm	Strongly positive - strongly hypersensitive to tuberculin protein	Suggests tuberculosis infection.

False negative tests may be caused by:

- miliary TB
- sarcoidosis
- HIV
- lymphoma
- very young age (e.g. < 6 months)

Heaf test

The Heaf test was previously used in the UK but has been since been discontinued. It involved injection of PPD equivalent to 100,000 units per ml to the skin over the flexor surface of the left forearm. It was then read 3-10 days later.

Diagnosis of active tuberculosis

Chest x-ray

- upper lobe cavitation is the classical finding of reactivated TB
- bilateral hilar lymphadenopathy

Sputum smear

- 3 specimens are needed
- rapid and inexpensive test
- stained for the presence of acid-fast bacilli (Ziehl-Neelsen stain)

    - all mycobacteria will stain positive (i.e. nontuberculous mycobacteria)
- the sensitivity is between 50-80%
    - this is decreased in individuals with HIV to around 20-30%

Sputum culture

- the gold standard investigation
    - more sensitive than a sputum smear and nucleic acid amplification tests
    - can assess drug sensitivities
- can take 1-3 weeks (if using liquid media, longer if solid media)

Nucleic acid amplification tests (NAAT)

- allows rapid diagnosis (within 24-48 hours)
- more sensitive than smear but less sensitive than culture

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2016 Tuberculosis guidelines

7 3

Suggest link Report broken link

Media



Tuberculosis

Osmosis - YouTube 2 1

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A 24-year-old lady presents 16 weeks pregnant. She complains of myalgia, sore throat and coryza and is noted to have a pyrexia of 38.1. She works in a nursing home which is currently closed due to an influenza A outbreak. On further questioning, you note that she has been vomiting every morning for the past 8 weeks and is no longer able to take oral medication. On examination her chest is clear but she is obviously coryzal with an erythematous oropharynx (no exudate). Chest X-ray is clear. Apart from informing the obstetrics team, how would you manage this patient?

- Admit, isolate and commence oseltamivir
- Admit and observe
- Oral antibiotics
- Admit, isolate and commence zanamivir
- Admit, isolate and await nose and throat swabs before commencing antiviral therapy

Submit answer

Reference ranges

Score: **22%**

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Admit, isolate and commence oseltamivir	33%
Admit and observe	11%
Oral antibiotics	1%
Admit, isolate and commence zanamivir	33%
Admit, isolate and await nose and throat swabs before commencing antiviral therapy	22%

This patient has had known exposure to influenza A. She should be admitted for observation and immediately commenced on empirical treatment. NICE recommends oseltamivir treatment as first line for suspected or confirmed influenza A in pregnancy. However, given that this patient has been vomiting, zanamivir (intranasal/intravenous) should be used in preference to oral Tamiflu (oseltamivir). Empirical treatment should be commenced immediately and not withheld until the results of swabs are available.

There may be a role for antibiotics if there is a co-existing infection or evidence of pneumonia on chest X-ray.

Discuss (9) | Improve

Next question

Antiviral agents

Drug	Mechanism of action	Indications	Adverse effects/toxicity
Aciclovir	Guanosine analog, phosphorylated by thymidine kinase which in	HSV, VZV	Crystalline nephropathy

Drug	Mechanism of action	Indications	Adverse effects/toxicity
	turn inhibits the viral DNA polymerase		
Ganciclovir	Guanosine analog, phosphorylated by thymidine kinase which in turn inhibits the viral DNA polymerase	CMV	Myelosuppression/agranulocytosis
Ribavirin	Guanosine analog which inhibits inosine monophosphate (IMP) dehydrogenase, interferes with the capping of viral mRNA	Chronic hepatitis C, RSV	Haemolytic anaemia
Amantadine	Inhibits uncoating (M2 protein) of virus in cell. Also releases dopamine from nerve endings	Influenza, Parkinson's disease	Confusion, ataxia, slurred speech
Oseltamivir	Inhibits neuraminidase	Influenza	
Foscarnet	Pyrophosphate analog which inhibits viiral DNA polymerase	CMV, HSV if not responding to aciclovir	Nephrotoxicity, hypocalcaemia, hypomagnasaemia, seizures
Interferon-α	Human glycoproteins which inhibit synthesis of mRNA	Chronic hepatitis B & C, hairy cell leukaemia	Flu-like symptoms, anorexia, myelosuppression
Cidofovir	Acyclic nucleoside phosphonate, and is therefore independent of phosphorylation by viral enzymes (compare and contrast with aciclovir/ganciclovir)	CMV retinitis in HIV	Nephrotoxicity

Anti-retroviral agent used in HIV

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), didanosine, lamivudine, stavudine, zalcitabine

Protease inhibitors (PI)

- inhibits a protease needed to make the virus able to survive outside the cell
- examples: indinavir, nelfinavir, ritonavir, saquinavir

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz



Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.8%**

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Back to top

A 35 year-old Brazilian accountant presented to the medical outpatient clinic with progressive leg swelling over the last 18 months. His mobility had become increasingly restricted due to both the swelling and shortness of breath on exertion. His father and paternal grandfather had both died from ischaemic heart disease in their 60s. His past medical history was unremarkable. He had smoked 15 cigarettes a day for 15 years and drank 15 units of alcohol per week.

On examination, his temperature was 36.5ºC, blood pressure was 95/50 mmHg and heart rate was 75 beats per minute. His JVP was elevated to the angle of the jaw. His chest was clear on auscultation, but a third heart sound and pan-systolic murmur were audible on auscultation of the precordium. Bilateral pitting oedema to mid-thigh level was present.

Investigations:

Haemoglobin	131 g/L (130-180)
White cell count	6.9 x 10^9/L (4.0-11.0)
Neutrophil count	3.1 x 10^9/L (2.0-7.5)
Lymphocyte count	1.9 x 10^9/L (1.3-3.5)
Eosinophil count	1.1 X 10^9/L (0.1-0.4)
Platelets	260 x 10^9/L (150-400)

Sodium	132 mmol/L (135-145)
Potassium	3.6 mmol/L (3.5-5.0)
Urea	8.0 mmol/L (2.5-7.5)
Creatinine	101 mol/L (20-90)
Fasting plasma glucose	7.2 mmol/L (3.0-6.0)

Echocardiogram	Moderately impaired left ventricular systolic function Dilated left ventricle with moderate mitral regurgitation

What is the most appropriate treatment for the underlying condition?

- Sulfadiazine and pyrimethamine
- Doxycycline
- Artesunate

Benznidazole

Mebendazole

Submit answer

Reference ranges

Score: **22%**

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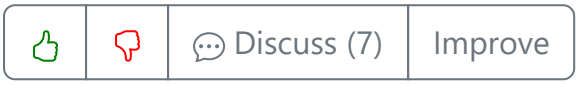
Echocardiogram	Moderately impaired left ventricular systolic function Dilated left ventricle with moderate mitral regurgitation

What is the most appropriate treatment for the underlying condition?

Sulfadiazine and pyrimethamine	13%
Doxycycline	15%
Artesunate	5%

Benznidazole	43%
Mebendazole	24%

This patient has developed chronic infection with tropical trypanosomiasis (Chagas disease). The parasite is endemic in South America, and is transmitted by the triatomine bug. Initial infection may present with transient fever and/or a swelling around the site of inoculation. In this case, chronic infection has developed; this may consist of dilated cardiomyopathy, arrhythmias or oesophageal dysmotility mimicking achalasia. Though this patient has some risk factors for ischaemic heart disease, the chances of this affecting him at this age are relatively small. The current treatment of choice for tropical trypanosomiasis is benznidazole.



Next question

Trypanosomiasis

Two main form of this protozoal disease are recognised - African trypanosomiasis (sleeping sickness) and American trypanosomiasis (Chagas' disease).

Two forms of **African trypanosomiasis**, or **sleeping sickness**, are seen - *Trypanosoma gambiense* in West Africa and *Trypanosoma rhodesiense* in East Africa. Both types are spread by the tsetse fly. *Trypanosoma rhodesiense* tends to follow a more acute course. Clinical features include:

- Trypanosoma chancre - painless subcutaneous nodule at site of infection
- intermittent fever
- enlargement of posterior cervical lymph nodes
- later: central nervous system involvement e.g. somnolence, headaches, mood changes, meningoencephalitis

Management

- early disease: IV pentamidine or suramin
- later disease or central nervous system involvement: IV melarsoprol

American trypanosomiasis, or **Chagas' disease**, is caused by the protozoan *Trypanosoma cruzi*. The vast majority of patients (95%) are asymptomatic in the acute phase although a chagoma (an erythematous nodule at site of infection) and periorbital oedema are sometimes seen. Chronic Chagas' disease mainly affects the heart and gastrointestinal tract

- myocarditis may lead to dilated cardiomyopathy (with apical atophy) and arrhythmias
- gastrointestinal features includes megaoesophagus and megacolon causing dysphagia and constipation

Management

- treatment is most effective in the acute phase using azole or nitroderivatives such as benznidazole or nifurtimox
- chronic disease management involves treating the complications e.g., heart failure

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

8 3

Chaga's disease

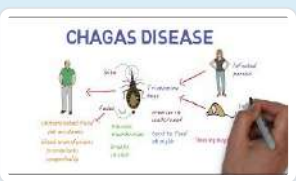
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Media



Sleeping Sickness - an introduction to African Trypanosomiasis

Let's Learn About Bugs - YouTube 7 0



Chagas Disease - An overview

Let's Learn About Bugs - YouTube 3 1



Chagas disease

Osmosis - YouTube 2 1

Report broken media

Score: **16.8%**

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A 65-year-old man is undergoing treatment in the intensive care unit. He had presented 8 days previously after suffering a seizure at home. He had been unable to give a coherent history at the time of presentation but his family had reported he had been unwell for about two weeks. Initial symptoms included a fever, myalgia, anorexia and an intense itching of his left arm. Subsequently, the patient had developed progressive confusion and agitation and was incoherent by the time of presentation to hospital. The patient was a long-term resident of the UK, but had made a trip back to his native India around 6 months previously. During that trip, his wife recalled the patient being bitten by a dog in his family village, although the wound had been minor and healed without incident after basic first aid. Medical history was otherwise fairly unremarkable, including hypertension, tablet-controlled type 2 diabetes and gout.

At presentation, the patient had been noted to been highly agitated and disorientated. Hypersalivation was noted and patient had been refusing to eat or drink. Examination had been limited by the patient's agitation but no overt focal neurological symptoms were noted in the cranial nerve or peripheral nervous examination. After the patient had been sedated, it was noted that striking a large muscle group with a tendon hammer led to few seconds of mounding of the muscle.

CSF protein	457 g / dL
CSF Microscopy	NAD
CSF rabies virus neutralising antibodies	Not detected
Serum rabies virus neutralising antibodies	Detected

At the present time, the patient is receiving end-of-life care. He is sedated and appears comfortable secondary to midazolam delivered by a syringe driver. His family wish to be present at his bedside.

What is the appropriate personal protection for healthcare workers and family members to use while in contact with the patient?

- High isolation
- Standard precautions
- Respiratory isolation
- Reverse isolation
- Biocontainment precautions

Submit answer

Reference ranges

Score: **22%**

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What is the appropriate personal protection for healthcare workers and family members to use while in contact with the patient?

High isolation	14%
Standard precautions	49%
Respiratory isolation	14%
Reverse isolation	5%
Biocontainment precautions	19%

The patient is unfortunately suffering from rabies. His presentation is consistent with the classical form of 'furious rabies', with fever, agitation, confusion and seizures. The response of the patient's muscles to being struck with a tendon hammer described in the question is called myoedema and is suggestive of rabies. Neutralising serum antibodies may be detected 7-8 days after the onset of symptoms but are only occasionally found in CSF. An alternative form of the disease ('paralytic rabies') is also known, featuring an ascending paralysis or symmetric quadriparesis.

The patient presumably contracted the disease on his previous trip to India when he was bitten by a dog. An incubation period of 6 months is not uncommon (mean incubation time 274 days in one study) with cases reported with incubation time up to 10 years. By the time neurological symptoms have taken hold, death is inevitable in unimmunised patients. This highlights the need for appropriate vaccination for individuals travelling to high-risk areas, as well as post-exposure vaccination and immunoglobulin administration.

Lyssaviruses such as rabies cannot cross intact skin and humans are regarded as an end-host (outside of transplantation-associated transmission). Therefore, only standard infection-prevention precautions such as gloves and gowns are required.

High isolation refers to the extreme precautions taken with high-transmissible and dangerous infections such as Ebola. Respiratory isolation requires the use of a mask to prevent droplet transmission by the respiratory route. Reverse isolation refers to techniques required to prevent external pathogens causing harm to an immunosuppressed patient. Biocontainment refers to laboratory biosafety techniques required when handling highly pathogenic organisms.

Crowcroft N, Thampi N. The prevention and management of rabies. BMJ 2015;350:g7827.

Discuss (3) Improve

Next question

Rabies

Rabies is a viral disease that causes an acute encephalitis. The rabies virus is classed as a RNA rhabdovirus (specifically a lyssavirus) and has a bullet-shaped capsid. The vast majority of cases are caused by dog bites but it may also be transmitted by bat, raccoon and skunk bites.

After entry, the virus replicates in muscle tissue near the site of the bite before entering the peripheral nervous system. It then travels up to the central nervous system, where it causes encephalitis, leading to severe neurological symptoms. The virus eventually reaches the salivary glands, facilitating transmission.

Rabies is estimated to still kill around 25,000-50,000 people across the world each year. The vast majority of the disease burden falls on people in poor rural areas of Africa and Asia. Children are

particularly at risk.

Features

- prodrome: headache, fever, agitation
- hydrophobia: water-provoking muscle spasms
- hypersalivation
- Negri bodies: cytoplasmic inclusion bodies found in infected neurons

There is now considered to be 'no risk' of developing rabies following an animal bite in the UK and the majority of developed countries. Following an animal bite in at-risk countries:

- the wound should be washed
- if an individual is already immunised then 2 further doses of vaccine should be given
- if not previously immunised then human rabies immunoglobulin (HRIG) should be given along with a full course of vaccination. If possible, the dose should be administered locally around the wound

If untreated the disease is nearly always fatal.

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Media



Rabies

macrophage - YouTube 2 0

Report broken media

Score: **16.8%**

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A 65 year-old gentleman with type 2 diabetes mellitus and alcoholism presents with fever, headache and neck stiffness. He has a history of previous anaphylactic reaction to penicillin.

On examination the temperature is 37.9ºC, respiratory rate is 20 breaths/min and heart rate is 105 beats per minute. Nutritional state and dental hygiene is poor. The chest is clear to auscultation. The abdomen is soft and non-tender. The Glasgow coma scale score is 15, the patient is uncomfortable during pen-torch pupillary examination and neck flexion is limited by pain.

CSF examination reveals:

White Cells	560 per mm^3 (85% polymorphs)
Red cells	8 per mm^3
Protein	0.9g/L
Glucose	3.3mmol/L (serum glucose 9.2mmol/L
Gram stain	Gram-negative coccobacilli

Which antimicrobial agent should be initiated?

- Vancomycin
- Ceftriaxone
- Chloramphenicol
- Rifampicin
- Gentamicin

Submit answer

Reference ranges

Score: **22%**

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Protein	0.9g/L
Glucose	3.3mmol/L (serum glucose 9.2mmol/L
Gram stain	Gram-negative coccobacilli

Which antimicrobial agent should be initiated?

Vancomycin	12%
Ceftriaxone	36%
Chloramphenicol	33%
Rifampicin	5%
Gentamicin	14%

This patient has meningitis caused by Haemophilus influenzae. Immunocompromised patients including those with alcoholism or diabetes are at risk.

Other gram -ve coccobacilli include Gardnerella vaginalis, Chlamydia trachomatis, Bordetella pertussis, Coxiella burnetti and Haemophilus ducreyi however these would not classically cause meningitis.

Cefotaxime or ceftriaxone would be the first-line treatment for haemophilus meningitis however for those with a history of anaphylaxis to penicillin, chloramphenicol is the most appropriate choice.

Chloramphenicol would also be the appropriate choice in meningococcal meningitis in a penicillin-allergic patient.

(Source: BNF)

Discuss (4) | Improve

Next question >

Meningitis: management of suspected bacterial meningitis

The investigation and management of suspected bacterial meningitis are usually intertwined given the potential negative impact of delayed antibiotic treatment.

All patients should be transferred to hospital urgently. If patients are in a pre-hospital setting (for example a GP surgery) and meningococcal disease is suspected then intramuscular benzylpenicillin may be given, as long as this doesn't delay transit to hospital.

An ABC approach should be taken initially

- Airway
- Breathing
- Circulation
- Disability: GCS, ; focal neurological signs; seizures; papilloedema;

Senior review if any **warning signs** are present (please see the Meningitis Research Foundation algorithm link for the full list). Examples include:

- rapidly progressive rash
- poor peripheral perfusion
- respiratory rate < 8 or > 30 / min *or* pulse rate < 40 or > 140 / min
- pH < 7.3 *or* WBC< 4 *10^9/L *or* lactate > 4 mmol/L
- GCS < 12 or a drop of 2 points
- poor response to fluid resuscitation

A key decision is when/whether to attempt a lumbar puncture (LP)

- LPs require an aseptic technique and are potentially technical difficult - this means they make take time and delay other treatment
- on the other hand, having a cerebrospinal fluid (CSF) sample can be very useful in confirming the diagnosis and guiding antibiotic treatment
- as always, patient safety means that if there is any doubt IV antibiotics should be given as a priority

Lumbar puncture should be delayed in the following circumstances

- signs of severe sepsis or a rapidly evolving rash
- severe respiratory/cardiac compromise
- significant bleeding risk
- signs of raised intracranial pressure
    - focal neurological signs
    - papilloedema
    - continuous or uncontrolled seizures
    - GCS ≤ 12

Management of patients without indication for delayed LP

- IV access → take bloods and blood cultures
- lumbar puncture
    - if this cannot be done within the first hour, IV antibiotics should be given after blood cultures have been taken
- IV antibiotics
    - 3 months - 50 years: BNF recommends cefotaxime (or ceftriaxone)
    - > 50 years: BNF recommends cefotaxime (or ceftriaxone) + amoxicillin (or ampicillin) for adults
- IV dexamethasone
    - the BNF recommend to '*consider adjunctive treatment with dexamethasone (particularly if pneumococcal meningitis suspected in adults), preferably starting before or with first dose of antibacterial, but no later than 12 hours after starting antibacterial; avoid dexamethasone in septic shock, meningococcal septicaemia, or if immunocompromised, or in meningitis following surgery*'
- CT scan is not normally indicated

Management of patients with signs of raised intracranial pressure

- get critical care input
- secure airway + high-flow oxygen
- IV access → take bloods and blood cultures
- IV dexamethasone
- IV antibiotics as above
- arrange neuroimaging

Management of patients with signs of severe sepsis or a rapidly evolving rash

- get critical care input
- secure airway + high-flow oxygen
- IV access → take bloods and blood cultures
- IV fluid resuscitation
- IV antibiotics as above

Investigations

The bloods that have already been taken should be tested for the following:

- full blood count
- renal function
- glucose
- lactate
- clotting profile
- CRP

If an LP has been performed, the CSF should be tested for:

- glucose, protein, microscopy and culture
- lactate
- meningococcal and pneumococcal PCR
- enteroviral, herpes simplex and varicella-zoster PCR
- consider investigations for TB meningitis

Additional tests that may be helpful include:

- blood gases
- throat swab for meningococcal culture

BNF antibiotic guidelines

Scenario	BNF recommendation
Initial empirical therapy aged < 3 months	IV cefotaxime + amoxicillin (or ampicillin)
Initial empirical therapy aged 3 months - 50 years	IV cefotaxime (or ceftriaxone)
Initial empirical therapy aged > 50 years	IV cefotaxime (or ceftriaxone) + amoxicillin (or ampicillin)
Meningococcal meningitis	IV benzylpenicillin or cefotaxime (or ceftriaxone)
Pneumococcal meningitis	IV cefotaxime (or ceftriaxone)
Meningitis caused by *Haemophilus influenzae*	IV cefotaxime (or ceftriaxone)
Meningitis caused by Listeria	IV amoxicillin (or ampicillin) + gentamicin

If the patient has a history of immediate hypersensitivity reaction to penicillin or to cephalosporins the BNF recommends using chloramphenicol.

Management of contacts

Prophylaxis needs to be offered to households and close contacts of patients affected with meningococcal meningitis. Prophylaxis should also be offered to people who have been exposed to respiratory secretion, regardless of the closeness of contact. The risk to contacts is highest in the first 7 days but persists for at least 4 weeks.

Management

- people who have been exposed to a patient with confirmed bacterial meningitis should be given prophylactic antibiotics if they have close contact within the 7 days before onset
- oral ciprofloxacin or rifampicin may be used. The Health Protection Agency (HPA) guidelines now state that whilst either may be used ciprofloxacin is the drug of choice as it is widely available and only requires one dose
- meningococcal vaccination should be offered to close contacts when serotype results are available, including booster doses to those who had the vaccine in infancy
- for pneumococcal meningitis, no prophylaxis is generally needed. There are however exceptions to this. If a cluster of cases of pneumococcal meningitis occurs the HPA have a protocol for offering close contacts antibiotic prophylaxis. Please see the link for more details



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Meningitis Research Foundation

22 13

Meningitis protocol

BNF

15 9

Medical emergencies in the community

Health Protection Agency 15 7

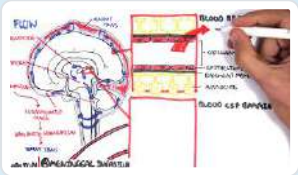
2018 Guidance for public health management of meningococcal disease in the UK

NICE 13 5

2010 Bacterial meningitis and meningococcal septicaemia guidelines

Suggest link Report broken link

Media



(Bacterial) Meningitis Pathophysiology

Armando Hasudungan - YouTube 5 0



Meningitis

Osmosis - YouTube 4 0

Report broken media

Score: **16.8%**

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A 22-year-old second generation British Pakistani is seen by the Tuberculosis (TB) Specialist Nurse as part of the contact tracing service. Her mother has recently been diagnosed with active TB after presenting to the respiratory clinic with a 3-month history of night sweats, weight loss, and haemoptysis.

The patient is clinically well and denies any focal symptomatology. Her past medical history is unremarkable and she is a non-smoker. She studies full-time at university but continues to live at home with her extended family. She was fully immunised as a child.

On examination, she is afebrile. Her respiratory rate is 16/min and her oxygen saturations are 97% breathing room air. There is no cervical lymphadenopathy, her chest is clear and her abdomen is soft and non-tender.

A Mantoux test is performed and read 72 hours later, at which time a 7mm area of induration is noted lying within a 16mm area of erythema. An interferon-gamma release assay (IGRA) is requested. What does a positive IGRA indicate?

Latent TB or prior immunisation	16%
Latent TB	38%
Prior immunisation	2%
Active or latent TB	38%
Active TB or prior immunisation	5%

The area of induration described in the vignette constitutes a positive Mantoux test. The area of surrounding erythema is not taken into account.

We are told that the patient has been fully immunised as a child and she may have therefore received the Bacille Calmette-Guerin (BCG) vaccination; a cause of a falsely positive Mantoux test. The other possibilities, in this case, are active or latent TB. An IGRA would be advantageous in this setting as prior BCG vaccination does not produce a false positive result. A positive test would, therefore, indicate prior exposure to M. tuberculosis.

The main disadvantage of the IGRA is its inability to distinguish between active and latent TB.

Tuberculosis: screening and diagnosis ★

Screening for latent tuberculosis

The Mantoux test is the main technique used to screen for latent tuberculosis. In recent years the interferon-gamma blood test has also been introduced. It is used in a number of specific situations such as:

- the Mantoux test is positive or equivocal
- people where a tuberculin test may be falsely negative (see below)

Mantoux test

- 0.1 ml of 1:1,000 purified protein derivative (PPD) injected intradermally
- result read 2-3 days later

Diameter of induration	Positivity	Interpretation
< 6mm	Negative - no significant hypersensitivity to tuberculin protein	Previously unvaccinated individuals may be given the BCG
6 - 15mm	Positive - hypersensitive to tuberculin protein	Should not be given BCG. May be due to previous TB infection or BCG
> 15mm	Strongly positive - strongly hypersensitive to tuberculin protein	Suggests tuberculosis infection.

False negative tests may be caused by:

- miliary TB
- sarcoidosis
- HIV
- lymphoma
- very young age (e.g. < 6 months)

Heaf test

The Heaf test was previously used in the UK but has been since been discontinued. It involved injection of PPD equivalent to 100,000 units per ml to the skin over the flexor surface of the left

forearm. It was then read 3-10 days later.

Diagnosis of active tuberculosis

Chest x-ray

- upper lobe cavitation is the classical finding of reactivated TB
- bilateral hilar lymphadenopathy

Sputum smear

- 3 specimens are needed
- rapid and inexpensive test
- stained for the presence of acid-fast bacilli (Ziehl-Neelsen stain)
    - all mycobacteria will stain positive (i.e. nontuberculous mycobacteria)
- the sensitivity is between 50-80%
    - this is decreased in individuals with HIV to around 20-30%

Sputum culture

- the gold standard investigation
    - more sensitive than a sputum smear and nucleic acid amplification tests
    - can assess drug sensitivities
- can take 1-3 weeks (if using liquid media, longer if solid media)

Nucleic acid amplification tests (NAAT)

- allows rapid diagnosis (within 24-48 hours)
- more sensitive than smear but less sensitive than culture

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

7 3

2016 Tuberculosis guidelines

Suggest link

Report broken link

Media



Tuberculosis

Osmosis - YouTube 2 1

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Score: **16.8%**

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A 54-year-old oil businessman who frequently visits the Gambia and is usually careful with his malaria prophylaxis has been admitted with general malaise and relapsing/remitting fevers which seem to occur every third day. He returned from the Gambia about one week ago and on this occasion, he did not take his malaria prophylaxis as he has never caught it before. He has no past medical history of note and no regular medication. Malarial parasites are seen on the thick and thin films and it is confirmed as *Plasmodium vivax* by the Malaria Reference Laboratory.

What is the most appropriate management according to current UK guidelines?

- Chloroquine
- Atovaquone-proguanil
- Chloroquine and primaquine
- Artemether with lumefantrine
- Intravenous artesunate + primaquine

Submit answer

Reference ranges

Score: **0%**

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What is the most appropriate management according to current UK guidelines?

Chloroquine	7%
Atovaquone-proguanil	6%
Chloroquine and primaquine	44%
Artemether with lumefantrine	22%
Intravenous artesunate + primaquine	20%

Plasmodium vivax is the second most commonly seen cause of malaria by UK statistics after *Plasmodium falciparum* malaria. It is characteristically a cause of benign tertian malaria where fevers occur every three days. Current UK guidelines (Health Protection Association(are that non-falciparum malaria should be treated with chloroquine but *P. vivax* and *P. ovale* are unusual in that dormant parasites (or hypnozoites) can exist within the liver.

The only currently available treatment for hypnozoites is primaquine and vivax malaria is more effectively treated with a higher dose than ovale (30mg). Therefore the correct answer is chloroquine and high-dose primaquine. Anyone commencing treatment with primaquine should have their glucose-6-phosphate dehydrogenase status tested as primaquine could precipitate severe haemolysis in deficient patients.

Artesunate, atovaquone-proguanil and artemether with lumefantrine are treatments for falciparum malaria.

Malaria: non-falciparum

The most common cause of non-falciparum malaria is *Plasmodium vivax*, with *Plasmodium ovale* and *Plasmodium malariae* accounting for the other cases. *Plasmodium vivax* is often found in Central America and the Indian Subcontinent whilst *Plasmodium ovale* typically comes from Africa.

Plasmodium knowlesi is another non-falciparum species which causes clinical pathology, found predominantly in South East Asia.

Features

- general features of malaria: fever, headache, splenomegaly
- *Plasmodium vivax/ovale*: cyclical fever every 48 hours. *Plasmodium malariae*: cyclical fever every 72 hours
- *Plasmodium malariae*: is associated with nephrotic syndrome.

Ovale and vivax malaria have a hypnozoite stage and may therefore relapse following treatment.

Treatment

- in areas which are known to be chloroquine-sensitive then WHO recommend either an artemisinin-based combination therapy (ACT) or chloroquine
- in areas which are known to be chloroquine-resistant an ACT should be used
- ACTs should be avoided in pregnant women
- patients with ovale or vivax malaria should be given primaquine following acute treatment with chloroquine to destroy liver hypnozoites and prevent relapse



Next question

Textbooks

High-yield textbook

Extended textbook

Links

WHO

9 1

Malaria treatment guidelines

Suggest link

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Media



Malaria

Osmosis - YouTube

4 0

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Score: **16.7%**

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A 35-year-old man presents to the genitourinary medicine clinic with a 1-week history of an ulcer on his penis. It is painless and he feels it might be increasing in size, though there are no other ulcers. He has never had any ulcers before.

He is HIV positive and remains sexually active with one regular male partner. They do not usually use condoms as he strictly adheres to his antiretroviral medication schedule and has an undetectable viral load.

He had gonorrhoea aged 18 which was treated with antibiotics at the time and he has no history of any other sexually transmitted infections.

He has no other past medical history and no drug allergies. He takes no medications other than his antiretrovirals.

On examination, he has a single shallow ulcer 1 x 2 cm on the dorsal surface of the penis with a small rim of surrounding erythema. He has no other rashes or lymphadenopathy. The systemic examination is otherwise unremarkable with no focal neurology.

Swabs from the ulcer are negative for herpes simplex virus 1 and 2.
Penile swabs are negative for chlamydia and gonorrhoea.

Syphilis serology result:
Enzyme immunoassay (EIA) +
Treponema Pallidum particle agglutination assay (TPPA) +
Rapid plasma reagin (RPR) -

What is the best immediate management option?

- Intramuscular penicillin G and check repeat syphilis serology
- Intramuscular penicillin G and check syphilis PCR
- Check syphilis PCR
- Oral doxycycline and check repeat syphilis serology
- Oral doxycycline and check syphilis PCR

Submit answer

Reference ranges

Score: **0%**

1 -

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Treponema Pallidum particle agglutination assay (TPPA) +
Rapid plasma reagin (RPR) -

What is the best immediate management option?

Intramuscular penicillin G and check repeat syphilis serology	32%
Intramuscular penicillin G and check syphilis PCR	42%
Check syphilis PCR	13%
Oral doxycycline and check repeat syphilis serology	6%
Oral doxycycline and check syphilis PCR	7%

This gentleman has a painless single penile ulcer which given negative testing for HSV, should be investigated for syphilis.

His syphilis serology shows a positive screening EIA and follow up TPPA (both treponemal-specific). However, his RPR titre is undetectable. This could indicate either past infection only or early acute infection without seroconversion on the RPR.

This gentleman is having unprotected intercourse and is HIV positive, so high risk for syphilis even if he gives a history of a single long-term partner. He also gives no history of past infection and so he should be treated for syphilis with antibiotics. Given he has no allergies, the first choice should penicillin G.

However, for clarification regarding diagnosis, he requires further testing, the gold standard being syphilis PCR.

References:
Public Health England. (2015). Syphilis Serology. UK Standards for Microbiology Investigations. V 44 Issue 2. https://www.gov.uk/uk-standards-for-microbiology-investigations-smi-quality-and-consistency-in-clinical-laboratories

Centres for Disease Control and Prevention. 2015 Sexually Transmitted Diseases Treatment Guidelines - Syphilis. 2015. Available online at: http://www.cdc.gov/std/tg2015/syphilis.htm (accessed 12 July 2015)

Discuss (6) Improve

Next question

Syphilis

Syphilis is a sexually transmitted infection caused by the spirochaete *Treponema pallidum*. Infection is characterised by primary, secondary and tertiary stages. The incubation period is between 9-90 days

Primary features

- chancre - painless ulcer at the site of sexual contact
- local non-tender lymphadenopathy
- often not seen in women (the lesion may be on the cervix)

Secondary features - occurs 6-10 weeks after primary infection

- systemic symptoms: fevers, lymphadenopathy
- rash on trunk, palms and soles
- buccal 'snail track' ulcers (30%)
- condylomata lata (painless, warty lesions on the genitalia)





Tertiary features

- gummas (granulomatous lesions of the skin and bones)
- ascending aortic aneurysms
- general paralysis of the insane
- tabes dorsalis
- Argyll-Robertson pupil

Features of congenital syphilis

- blunted upper incisor teeth (Hutchinson's teeth), 'mulberry' molars
- rhagades (linear scars at the angle of the mouth)
- keratitis
- saber shins
- saddle nose
- deafness

AI + Q 123

Next question >

B I A

Textbooks

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Extended textbook

Links

DermIS.net 5 5

Picture of condylomata lata

Clinical Knowledge Summaries 8 4

Syphilis guidelines

DermNet NZ 12 5

Syphilis images

Suggest link Report broken link

Media



Syphilis

Osmosis - YouTube 5 0

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A 35-year-old female returned from Tanzania 2 weeks ago. She had consulted her GP after feeling unwell over the previous 3 days with intermittent symptoms of fever, headache and generalised arthralgia. She had taken doxycycline as an antimalarial and denies missing any of the prescribed doses and had been fully vaccinated prior to her travel.

She is referred to the medical take where you retake the history. She admits to getting mosquito bites regularly whilst away, one of them she seemed to react severely to and this has left a mark on her shoulder.

On examination she appears drowsy, she has mild heptosplenomegally and there is a well circumscribed, rubbery, painful, red lesion approximately 5cm in diameter above her left shoulder.

What is the likely diagnosis?

- *Plasmodium falciparum*
- *Trypanosomiasis brucei rhodesiensi*
- *Trypanosomiasis brucei cruzi*
- *Trypanosomiasis brucei gambiensi*
- *Toxoplasmosis*

Submit answer

Reference ranges

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A 35-year-old female returned from Tanzania 2 weeks ago. She had consulted her GP after feeling unwell over the previous 3 days with intermittent symptoms of fever, headache and generalised arthralgia. She had taken doxycycline as an antimalarial and denies missing any of the prescribed doses and had been fully vaccinated prior to her travel.

She is referred to the medical take where you retake the history. She admits to getting mosquito bites regularly whilst away, one of them she seemed to react severely to and this has left a mark on her shoulder.

On examination she appears drowsy, she has mild heptosplenomegally and there is a well circumscribed, rubbery, painful, red lesion approximately 5cm in diameter above her left shoulder.

What is the likely diagnosis?

Plasmodium falciparum	5%
Trypanosomiasis brucei rhodesiensi	43%
Trypanosomiasis brucei cruzi	16%
Trypanosomiasis brucei gambiensi	35%
Toxoplasmosis	1%

Human African trypanosomiasis (HAT), also known as sleeping sickness, is caused by protozoan parasites. There are two forms of the disease, an acute form occurring mainly in East Africa caused by *Trypanosoma brucei rhodesiense*, and a more chronic form occurring in West and Central Africa, caused by *Trypanosoma brucei gambiense*. *Trypanosomiasis brucei cruzi* is endemic in south america where it causes Chagas disease.

Trypanosomiasis rhodesiense is transmitted by the tsetse fly which are classically found on safari in Tanzania. *T. rhodesiense* will often form a chancre at the site of inoculation and can often be the first and only sign of disease where it is rapidly progressive.

Given the story we are immediately highlighted to the potential of malaria. However given that she has taken adequate prophylaxis we should keep an open mind to other potential diagnoses. Furthermore her illness started 3 days prior to her admission to hospital making *P.falciparum* less likely as this would have resulted in her being very unwell by this point.

As explained *T.Cruzi* is endemic in south america and can therefore be discounted early on. *T.gambiense* is likely to present with a much more chronic picture. Toxoplasmosis is very unlikely in an immunocompetant host and her travel to Tanzania would not necessarily increase her risk of acquiring the condition. As a result the most likely diagnosis is *T.rhodesiense*. Diagnosis can be made by demonstration of the parasite in the blood, lymph node/chancre aspirate or on CSF examination. The condition is fatal in the absence of treatment.

Discuss (8) | Improve

Next question >

Trypanosomiasis

Two main form of this protozoal disease are recognised - African trypanosomiasis (sleeping sickness) and American trypanosomiasis (Chagas' disease).

Two forms of **African trypanosomiasis**, or **sleeping sickness**, are seen - *Trypanosoma gambiense* in West Africa and *Trypanosoma rhodesiense* in East Africa. Both types are spread by the tsetse fly. *Trypanosoma rhodesiense* tends to follow a more acute course. Clinical features include:

- Trypanosoma chancre - painless subcutaneous nodule at site of infection
- intermittent fever
- enlargement of posterior cervical lymph nodes
- later: central nervous system involvement e.g. somnolence, headaches, mood changes, meningoencephalitis

Management

- early disease: IV pentamidine or suramin
- later disease or central nervous system involvement: IV melarsoprol

American trypanosomiasis, or **Chagas' disease**, is caused by the protozoan *Trypanosoma cruzi*. The vast majority of patients (95%) are asymptomatic in the acute phase although a chagoma (an erythematous nodule at site of infection) and periorbital oedema are sometimes seen. Chronic Chagas' disease mainly affects the heart and gastrointestinal tract

- myocarditis may lead to dilated cardiomyopathy (with apical atophy) and arrhythmias
- gastrointestinal features includes megaoesophagus and megacolon causing dysphagia and constipation

Management

- treatment is most effective in the acute phase using azole or nitroderivatives such as benznidazole or nifurtimox
- chronic disease management involves treating the complications e.g., heart failure

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

8 3

Chaga's disease

Suggest link

Report broken link

Media



Sleeping Sickness - an introduction to African Trypanosomiasis

Let's Learn About Bugs - YouTube

7 0



Chagas Disease - An overview

Let's Learn About Bugs - YouTube

3 1



Chagas disease

Osmosis - YouTube

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A 59 year-old man attends his general practitioner with a 10 day history of watery diarrhoea. His stools are unformed, sometimes associated with fresh red blood. He also has associated abdominal cramps for which he has been taking paracetamol. He estimates a 5kg weight loss during this period, and has struggled to tolerate food or fluids due to significant pain on swallowing. He also has an intermittent sensation of needing to defecate, but being unable to.

He has been diagnosed with HIV 8 years ago, and admits that he has not been attending follow up, or taking his medications. He is unsure what his last CD4 cell count was. He has had 5 episodes of pneumonia over the last 12 months, the last one was 4 months ago.

On examination, he has dry mucous membranes with reduced skin turgor. He has generalised abdominal tenderness, but no features of peritonism, or organomegaly.

Observations

Respiratory rate- 18 breaths per minute
Heart rate- 110 beats per minute
Blood pressure- 104/60 mmHg
Temperature 37.7ºC

What is the most likely pathogen causing his symptoms?

- Cryptosporidiosis
- Clostridium difficile
- Giardia
- E Coli 0157
- Cytomegalovirus colitis

Submit answer

Reference ranges

Score: **0%**

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A 59 year-old man attends his general practitioner with a 10 day history of watery diarrhoea. His stools are unformed, sometimes associated with fresh red blood. He also has associated abdominal cramps for which he has been taking paracetamol. He estimates a 5kg weight loss during this period, and has struggled to tolerate food or fluids due to significant pain on swallowing. He also has an intermittent sensation of needing to defecate, but being unable to.

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Observations

Respiratory rate- 18 breaths per minute
Heart rate- 110 beats per minute
Blood pressure- 104/60 mmHg
Temperature 37.7ºC

What is the most likely pathogen causing his symptoms?

Cryptosporidiosis	56%
Clostridium difficile	2%
Giardia	3%
E Coli 0157	4%
Cytomegalovirus colitis	35%

Cytomegalovirus (CMV) colitis is usually seen in patients who are immunocompromised. It is a member of the herpes virus, for which around 50-80% of the general population are seropositive. The virus lays dormant until the patient is sufficiently immunocompromised.

Approximately 30% of all patients with AIDS will develop a gastrointestinal CMV infection. This usually occurs when the CD4+ counts drop to below 100 mm3.

CMV can affect the gastrointestinal tract from the oesophagus to the rectum, and symptoms

depend on the site of infection. CMV oesophagitis causes odynophagia and retrosternal chest pain. CMV enterocolitis commonly presents with diarrhoea, fever, weight loss and per rectal bleeding. Tenesmus can occur if there is rectal involvement.

Cryptosporidiosis is a protozoal infection caused by cryptosporidium parvum. Immunocompromised individuals are particularly susceptible. The incubation period is between 2-10 days (average 7) and symptoms usually last about 1 to 2 weeks, although can occur for longer. The most common symptom is watery diarrhoea. Other symptoms include stomach cramps, dehydration, nausea and vomiting and fever.

E Coli 0157 is a relatively rare cause of infective diarrhoea in England. Symptoms range from mild gastroenteritis through to severe bloody diarrhoea. It can be associated with haemolytic uraemic syndrome and thrombotic thrombocytopaenic purpura.

Giardia is causes by the protozoon parasite Giardia lamblia. Symptoms include diarrhoea, foul smelling flatulence and abdominal cramps. Symptoms typically last between 2-4 weeks, but chronic diarrhoea and malabsorption can occur.

The patient has likely got a very low CD4+ count, due to his non-compliance with medication and follow up and history of recurrent pneumonia. CMV colitis with rectal and oesophageal involvement would explain the tenesmus, odynophagia, along with the bloody diarrhoea, abdominal cramps and weight loss.

Cryptosporidiosis and giardia do not tend to cause bloody diarrhoea. Clostridium difficile is an important cause of diarrhoea in immunocompromised patients. However, neither clostridium difficule nor E Coli 0157 this would not explain the odynophagia or tenesmus.

👍 👎 💬 Discuss (5) Improve

Next question ›

Cytomegalovirus ★

Cytomegalovirus (CMV) is one of the herpes viruses. It is thought that around 50% of people have been exposed to the CMV virus although it only usually causes disease in the immunocompromised, for example people with HIV or those on immunosuppressants following organ transplantation.

Pathophysiology

- infected cells have a 'Owl's eye' appearance due to intranuclear inclusion bodies

Patterns of disease

Congenital CMV infection

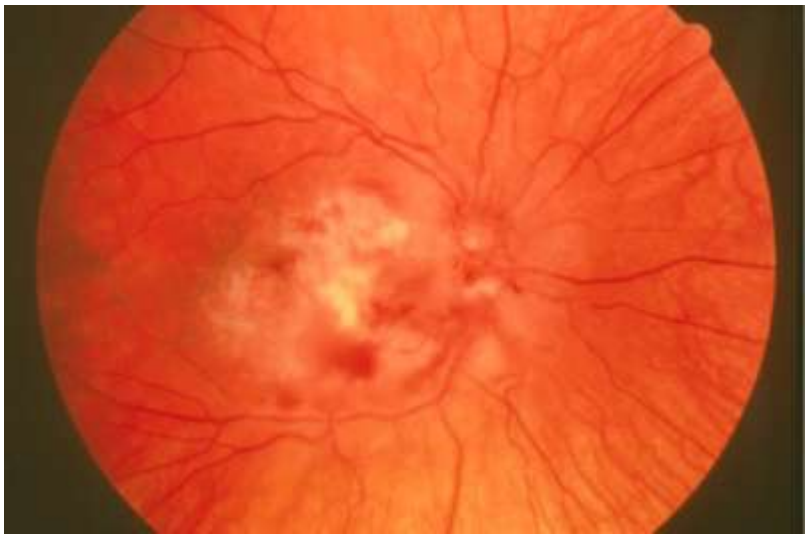
- features include growth retardation, pinpoint petechial 'blueberry muffin' skin lesions, microcephaly, sensorineural deafness, encephalitiis (seizures) and hepatosplenomegaly

CMV mononucleosis

- infectious mononucelosis-like illness
- may develop in immunocompetent individuals

CMV retinitis

- common in HIV patients with a low CD4 count (< 50)
- presents with visual impairment e.g. 'blurred vision'. Fundoscopy shows retinal haemorrhages and necrosis, often called 'pizza' retina
- IV ganciclovir is the treatment of choice



Fundus photograph showing CMV retinitis. Credit: National Eye Institute, National Institutes of Health

CMV encephalopathy

- seen in patients with HIV who have low CD4 counts

CMV pneumonitis

CMV colitis

123

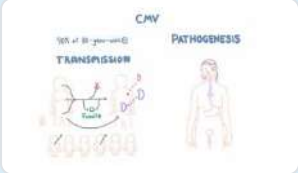
Next question

Textbooks

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Media



CMV - Mono, Congenital Infection, Immunocompromised Hosts

macrophage - YouTube 4 0



CMV infection after transplant

Osmosis - YouTube 4 2

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Score: **16.7%**

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A 47-year-old man presented with a headache to the emergency department a few days ago. His partner accompanied him and reported that he has been more confused over the last few days. He had also been febrile and complained of swollen glands in his neck. He had a self-terminating seizure whilst awaiting transfer to the medical ward.

His past medical history is significant for HIV. His partner reported he is not compliant with his anti-retroviral treatment and last year was hospitalised for severe pneumonia.

A CT head showed multiple ring-enhancing lesions. The infectious diseases team reviewed him and suggested additional blood tests, results of which are shown below:

Hb	140 g/L	(135-180)
Platelets	167 * 10^9/L	(150 - 400)
WBC	3.8 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	4.5 mmol/L	(3.5 - 5.0)
Urea	8.5 mmol/L	(2.0 - 7.0)
Creatinine	137 µmol/L	(55 - 120)
CRP	50 mg/L	(< 5)
Toxoplasmosis serology	positive	
Cryptococcal serology	negative	
β-d-Glucan	normal	
Galactomannan	normal	

What is the most appropriate treatment to give this patient?

- Co-trimoxazole and prednisolone
- Fluconazole
- Pyrimethamine and sulphadiazine
- Amphotericin B
- Pentamidine and prednisolone

Submit answer

Reference ranges

Score: **0%**

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CRP	50 mg/L	(< 5)
Toxoplasmosis serology	positive	
Cryptococcal serology	negative	
β-d-Glucan	normal	
Galactomannan	normal	

What is the most appropriate treatment to give this patient?

Co-trimoxazole and prednisolone	5%
Fluconazole	0%
Pyrimethamine and sulphadiazine	**90%**
Amphotericin B	2%
Pentamidine and prednisolone	2%

Immunocompromised patients with toxoplasmosis are treated with pyrimethamine plus sulphadiazine

Important for me Less important

This man has a background of HIV and is not compliant with treatment, therefore is immunocompromised and at risk of opportunistic infections. Toxoplasmosis serology is positive and the multiple ring-enhancing lesions on CT support this diagnosis. Management of toxoplasmosis in immunocompromised patients is with pyrimethamine and sulphadiazine.

Co-trimoxazole and prednisolone is used in the management of *Pneumocystis jirovecii* pneumonia. There are no chest symptoms to suggest this is the cause of this patient's presentation.

Fluconazole can be used to treat fungal infections such as candidiasis.

Amphotericin B is used in the management of cryptococcal infection. Cryptococcus serology is negative, and the CT head would show cerebral oedema and meningeal enhancement if this was the diagnosis.

Pentamidine and prednisolone is an alternative treatment regimen for *Pneumocystis jirovecii* pneumonia.

Discuss Improve

Next question

Toxoplasmosis

Toxoplasma gondii is an obligate intracellular protozoan that infects the body via the gastrointestinal tract, lung or broken skin. It's oocysts release trophozoites which migrate widely around the body including to the eye, brain and muscle. The usual animal reservoir is the cat, although other animals such as rats carry the disease.

Immunocompetent patients

Most infections are asymptomatic. Symptomatic patients usually have a self-limiting infection, often having clinical features resembling infectious mononucleosis (fever, malaise, lymphadenopathy). Other less common manifestations include meningoencephalitis and myocarditis.

Serology is the investigation of choice.

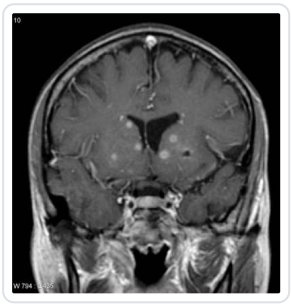
No treatment is usually required unless the patient has a severe infection or is immunosuppressed.

HIV/immunosuppressed patients

Cerebral toxoplasmosis accounts for around 50% of cerebral lesions in patients with HIV

- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring-enhancing lesions, mass effect may be seen
- management: pyrimethamine plus sulphadiazine for at least 6 weeks





Immunosuppressed patients may also develop a chorioretinitis secondary to toxoplasmosis.

Congenital toxoplasmosis

Congenital toxoplasmosis is due to transplacental spread from the mother. It causes a variety of effects to the unborn child including

- neurological damage
  - cerebral calcification
  - hydrocephalus
  - chorioretinitis
- ophthalmic damage
  - retinopathy
  - cataracts

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 28-year-old man presents to the clinic with a rash on his foot. He states that he first noticed it two weeks after returning from Thailand. The rash is very itchy and has been keeping him awake at night. He has no past medical history and takes no regular medicines. On examination he has a erythematous, oedematous, serpiginous track on the superior aspect of his right foot.

What is the treatment of choice for this condition?

- Clotrimazole
- Terbinafine
- Albendazole
- Flucloxacillin
- Doxycycline

Submit answer

Reference ranges

Score: **0%**

1 -
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What is the treatment of choice for this condition?

Clotrimazole	2%
Terbinafine	7%
Albendazole	80%
Flucloxacillin	1%
Doxycycline	9%

Albendazole is the treatment of choice for cutaneous larva migrans

Important for me Less important

The clinical history is suggestive of cutaneous larva migrans which is caused by infection of the dog hookworm Ancylostoma braziliense. Management is with albendazole or ivermectin.

Flucloxacillin and doxycycline are indicated for cellulitis.

Clotrimazole and terbinafine are used in the treatment of fungal infections such as ringworm.

Discuss (2) Improve

Next question

Cutaneous larva migrans

Cutaneous larva migrans is a dermatological condition prevalent in tropical and subtropical regions, largely attributable to cutaneous penetration and subsequent migration of nematode

larvae, primarily from the *Ancylostoma* genus (e.g. *Ancyclostoma braziliense*). Typically, the transmission vectors are faecal-contaminated soil or sand, posing significant risks to individuals with a history of barefoot beach visits or direct soil contact.

The clinical presentation typically involves an intensely pruritic, 'creeping', serpiginous, erythematous cutaneous eruption that advances over time. Symptoms can last for weeks to months, potentially leading to secondary bacterial infection due to excessive scratching. Diagnosis is typically clinical, based on exposure history and characteristic skin manifestations.



Treatment options revolve around anthelmintic agents, such as ivermectin or albendazole. Topical therapy with thiabendazole can also be effective, although it's generally less preferred due to a lower efficacy rate and higher side effect profile.

Prevention strategies largely involve patient education about appropriate protective measures, particularly avoiding direct skin contact with potentially contaminated soil. Given the zoonotic nature of cutaneous larva migrans, public health measures for animal defecation control can also contribute to reducing the prevalence of this condition.

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Center for Disease Control

Cutaneous Larva Migrans

3 2

Suggest link Report broken link

Score: **16.7%**

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A 33-year-old man who is HIV positive is admitted to the Emergency Department with confusion and drowsiness. He has been complaining of headaches for a number of days. On examination heart rate is 90/min, blood pressure 104/78 mmHg and temperature is 37.2ºC. He is confused giving a Glasgow Coma Scale (GCS) score of 14. There is no photophobia or neck stiffness.

His infectious diseases consultant reports that he is prescribed highly active antiretroviral treatment (HAART) but his compliance is poor and he often misses clinic appointments.

A CT scan is requested:

.



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What is the most likely diagnosis?

- Cerebral toxoplasmosis
- Tuberculosis
- CMV encephalitis
- Progressive multifocal leukoencephalopathy

Cryptococcal infection

Submit answer

Reference ranges

Score: **0%**

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A 33-year-old man who is HIV positive is admitted to the Emergency Department with confusion and drowsiness. He has been complaining of headaches for a number of days. On examination heart rate is 90/min, blood pressure 104/78 mmHg and temperature is 37.2ºC. He is confused giving a Glasgow Coma Scale (GCS) score of 14. There is no photophobia or neck stiffness.

His infectious diseases consultant reports that he is prescribed highly active antiretroviral treatment (HAART) but his compliance is poor and he often misses clinic appointments.

A CT scan is requested:

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© Image used on license from Radiopaedia

What is the most likely diagnosis?

Cerebral toxoplasmosis	50%
Tuberculosis	2%
CMV encephalitis	7%
Progressive multifocal leukoencephalopathy	34%

Cryptococcal infection 7%

HIV, neuro symptoms, multiple brain lesions with ring enhancement - toxoplasmosis

Important for me Less important

The CT scan shows multiple ring enhancing lesions consistent with cerebral toxoplasmosis. He is at risk of this due to his poor compliance with medication.

Cerebral toxoplasmosis is the most common neurological infection seen in HIV, occurring in up to 10% of patients.

Discuss (4) Improve

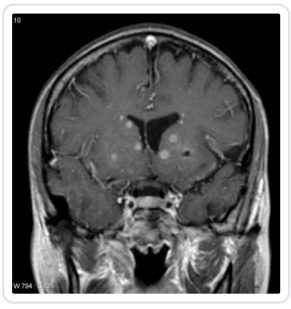
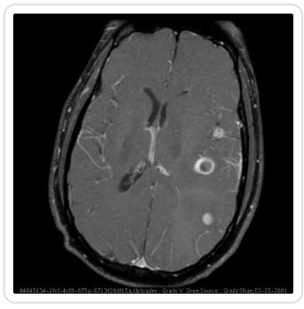
Next question

HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

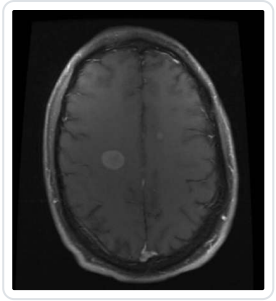
- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination

- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link Report broken link

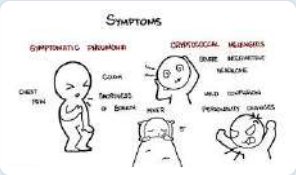
Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

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Score: **16.7%**

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A 25-year-old man presents to the genitourinary medicine clinic with a penile lesion which has been present for the last three weeks but is now starting to reduce in size. He has had two sexual partners in the last 2 months, who were both men. There is no history of travel outside of the UK in the last 3 years.

The lesion is a shallow ulcer with well-defined margins, which is not tender to palpation. He has some non-tender enlarged lymph nodes on both sides in the inguinal region.

What is the most likely diagnosis?

- Chancroid
- Donovanosis
- Lymphogranuloma venereum
- M-pox
- Primary syphilis

Submit answer

Reference ranges

Score: **0%**

1 -
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The lesion is a shallow ulcer with well-defined margins, which is not tender to palpation. He has some non-tender enlarged lymph nodes on both sides in the inguinal region.

What is the most likely diagnosis?

Chancroid	11%
Donovanosis	3%
Lymphogranuloma venereum	29%
M-pox	1%
Primary syphilis	57%

Primary syphilis is associated with **painless** ulceration

Important for me Less important

The correct answer is **primary syphilis** , which is caused by *Treponema pallidum*. This classically presents with a chancre at the site of entry of infection. This usually starts as a painless papule, which evolves into a painless ulcer. The lesion is usually single but multiple lesions can occur. It generally heals over a few weeks.

Chancroid is incorrect. This is a sexually transmitted disease caused by *Haemophilus ducreyi*. However, unlike in this patient, the ulcers are commonly multiple and very painful. The inguinal lymphadenopathy in chancroid is also generally painful. Chancroid is rare in the UK, and most cases are acquired abroad in Africa or South East Asia.

Donovanosis is incorrect. This condition is also called granuloma inguinale and is caused by *Klebsiella granulomatis*. The genital ulcers in this condition are painless with a classically 'beefy red' base. This diagnosis is less likely in this patient because the ulcers would not start to heal without treatment but would continue to enlarge, and because it is rarely seen in the UK. It is most commonly seen in tropical regions.

Lymphogranuloma venereum is incorrect. This is caused by serovars L1, L2 and L3 of *Chlamydia trachomatis* . It is more common in tropical areas but does occur in the UK, mainly in men who have sex with men. It can cause painless ulceration in the primary stage, but unlike in this scenario, the ulcers are small and heal within 1-3 days so this stage is generally not noticed by patients. The secondary stage is characterised by painful swollen inguinal lymph nodes or anal symptoms in patients who have acquired an anal/rectal infection.

M-pox is incorrect. The M-pox virus classically causes painful vesicular lesions, which usually start at the site of inoculation and are generally multiple. The lesions may then spread to involve other areas of the body.

Discuss (4) Improve

Next question

Syphilis

Syphilis is a sexually transmitted infection caused by the spirochaete *Treponema pallidum*. Infection is characterised by primary, secondary and tertiary stages. The incubation period is between 9-90 days

Primary features

- chancre - painless ulcer at the site of sexual contact
- local non-tender lymphadenopathy
- often not seen in women (the lesion may be on the cervix)

Secondary features - occurs 6-10 weeks after primary infection

- systemic symptoms: fevers, lymphadenopathy
- rash on trunk, palms and soles
- buccal 'snail track' ulcers (30%)
- condylomata lata (painless, warty lesions on the genitalia)





Tertiary features

- gummas (granulomatous lesions of the skin and bones)
- ascending aortic aneurysms
- general paralysis of the insane

- tabes dorsalis
- Argyll-Robertson pupil

Features of congenital syphilis

- blunted upper incisor teeth (Hutchinson's teeth), 'mulberry' molars
- rhagades (linear scars at the angle of the mouth)
- keratitis
- saber shins
- saddle nose
- deafness

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermIS.net 5 5

Picture of condylomata lata

Clinical Knowledge Summaries 8 4

Syphilis guidelines

DermNet NZ 12 5

Syphilis images

Suggest link Report broken link

Media



Syphilis

Osmosis - YouTube 5 0

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Score: **16.7%**

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A 35-year-old man presents to his general practitioner with two months of dysphagia and odynophagia. He has a past medical history of HIV. He is not compliant with anti-retroviral treatment.

Observations:

- Heart rate 88 beats per minute
- Blood pressure 120/72 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 98% on room air
- Temperature 37ºC

On examination, there are white patches on the gums, tongue and extending past the pharynx. The examination is otherwise unremarkable.

What is the most appropriate treatment?

- Amphotericin
- Fluconazole
- Lansoprazole
- Nystatin
- Surgical excision

Submit answer

Reference ranges

Score: **0%**

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- Respiratory rate 18/minute
- Oxygen saturations 98% on room air
- Temperature 37ºC

On examination, there are white patches on the gums, tongue and extending past the pharynx. The examination is otherwise unremarkable.

What is the most appropriate treatment?

Amphotericin	5%
Fluconazole	74%
Lansoprazole	1%
Nystatin	19%
Surgical excision	2%

Candidiasis in immunocompromised patients should be treated using high dose fluconazole

Important for me Less important

Fluconazole is the correct answer. The description of the lesions is typical of candidiasis. Given the patient has oesophageal symptoms in the form of dysphagia and odynophagia, we can assume that the oesophagus is involved, in addition to the oro-pharynx. The patient is not compliant with treatment for HIV and is therefore at risk of developing AIDS. Oesophageal candidiasis is an AIDS-defining illness. In immunocompromised patients, the treatment of candidiasis is with high dose fluconazole.

Amphotericin is incorrect. This is an intravenous treatment associated with significant toxicity. It is reserved for life-threatening, disseminated fungal infections.

Lansoprazole is incorrect. The dysphagia and odynophagia are not likely a result of gastro-oesophageal reflux.

Nystatin is incorrect. This would be suitable to treat oral thrush in an immunocompetent patient but would not be sufficient in this case.

Surgical excision is incorrect. A differential for a single white patch in the mouth or tongue might be oral leucoplakia, which is a pre-malignant condition and which could be surgically excised. However, in this context, multiple white patches in multiple locations is more likely to reflect candida.

Discuss | Improve

Next question ›

HIV: oesophageal candidiasis ★

Oesophageal candidiasis is the most common cause of oesophagitis in patients with HIV. It is generally seen in patients with a CD4 count of less than 100. Typical symptoms include dysphagia and odynophagia. Fluconazole and itraconazole are first-line treatments.

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media

Candidal infections

Osmosis - YouTube 2 1

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Score: **16.7%**

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A 65-year-old man presents to the emergency department complaining of a 1 day history of fever, new rash and severe pain in his leg. His past medical history includes type 2 diabetes and peripheral vascular disease. His medications include metformin and clopidogrel. He smokes twenty cigarettes daily and lives alone.

His observations are heart rate 110 beats per minute, blood pressure 88/51 mmHg, respiratory rate 21/minute, oxygen saturations 96% on room air and temperature 38.4ºC.

On examination, he appears unwell. He is clammy and diaphoretic. There is an area of erythema extending from his right calf to his right thigh. It is warm to touch and in the region of the thigh, is diffusely tender. The degree of tenderness seems out of proportion to the severity of the rash. Cardiovascular, respiratory and abdominal examinations are unremarkable.

Blood tests:

Hb	111 g/L	Male: (135-180) Female: (115 - 160)
Platelets	444 * 10^9/L	(150 - 400)
WBC	14.4 * 10^9/L	(4.0 - 11.0)
Na^+	131 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	9.8 mmol/L	(2.0 - 7.0)
Creatinine	180 µmol/L	(55 - 120)
CRP	176 mg/L	(< 5)
Lactate	3.4 mmol/L	(0.5 - 2.2)
Creatine kinase	1200 U/L	(40-320)

Despite antibiotics and aggressive fluid resuscitation with 3 litres of normal saline, he remains hypotensive with a blood pressure of 85/52 mmHg.

Given the likely diagnosis, what combination of organisms is likely to be responsible?

- *Bacteroides fragilis* and *Clostridium perfringens*
- *Pseudomonas aeruginosa* and *Escherichia coli*

- *Staphylococcus aureus* , *Streptococcus pyogenes* and *Enterococcus faecalis*
- *Streptococcus pyogenes* , *Escherichia coli* and *Clostridium perfringens*
- *Vibrio vulnificus* and *Pseudomonas aeruginosa*

Submit answer

Reference ranges

Score: **0%**

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Creatine kinase	1200 U/L	(40-320)

Despite antibiotics and aggressive fluid resuscitation with 3 litres of normal saline, he remains hypotensive with a blood pressure of 85/52 mmHg.

Given the likely diagnosis, what combination of organisms is likely to be responsible?

Bacteroides fragilis and *Clostridium perfringens*	15%
Pseudomonas aeruginosa and *Escherichia coli*	2%

Staphylococcus aureus , *Streptococcus pyogenes* and *Enterococcus faecalis*	36%
Streptococcus pyogenes , *Escherichia coli* and *Clostridium perfringens*	45%
Vibrio vulnificus and *Pseudomonas aeruginosa*	2%

Type 1, or polymicrobial, necrotising fasciitis is the most common

Important for me Less important

Streptococcus pyogenes , *Escherichia coli* and *Clostridium perfringens* is the correct answer. The diagnosis is necrotizing fasciitis, as evidenced by a severe soft tissue infection with pain and tenderness out of proportion to the clinical signs. Further supportive of evidence of necrotizing fasciitis versus a simple skin and soft tissue infection is the associated evidence of septic shock and organ dysfunction (acute kidney injury and raised lactate) with a raised creatine kinase. The most common type of necrotizing fasciitis is 'Type 1', which is caused by a combination of gram positive, gram negative and anaerobic bacteria. Furthermore, this type is more common in an older population with type 2 diabetes and peripheral vascular disease. It is the only option listed that is polymicrobial. Management is with antibiotics and aggressive surgical debridement.

Pseudomonas aeruginosa and *Escherichia coli* is the wrong answer. While both of these organisms can cause necrotizing fasciitis, they are both gram negative rods. They are less likely to cause necrotizing fasciitis either alone or in combination when compared to a polymicrobial combination.

Staphylococcus aureus , *Streptococcus pyogenes* and *Enterococcus faecalis* is incorrect. All of these organisms can cause necrotizing fasciitis. However, they are a less likely combination than the polymicrobial subtype and are all gram positive cocci. Type 2 necrotizing fasciitis tends to be caused by *Streptococcus pyogenes* alone and is more common in a younger population.

Bacteroides fragilis and *Clostridium perfringens* is the wrong answer. These are anaerobic bacteria that can cause necrotizing fasciitis but they are less likely to cause necrotizing fasciitis either alone or in combination than the polymicrobial subtype.

Vibrio vulnificus and *Pseudomonas aeruginosa* is the wrong answer. These are both gram negative rods rather than a polymicrobial combination. *Vibrio vulnificus* has been implicated in the development of necrotizing fasciitis and is a bacterium found in saltwater but this is a rare cause.

Discuss (3) Improve

Next question ›

Necrotising fasciitis

Necrotising fasciitis is a medical emergency that is difficult to recognise in the early stages.

It can be classified according to the causative organism:

- type 1 is caused by mixed anaerobes and aerobes (often occurs post-surgery in diabetics). This is the most common type
- type 2 is caused by *Streptococcus pyogenes*

Risk factors

- skin factors: recent trauma, burns or soft tissue infections
- diabetes mellitus
  - the most common preexisting medical condition
  - particularly if the patient is treated with SGLT-2 inhibitors
- intravenous drug use
- immunosuppression

The most commonly affected site is the perineum (Fournier's gangrene).

Features

- acute onset
- pain, swelling, erythema at the affected site
  - often presents as rapidly worsening cellulitis with pain out of keeping with physical features
  - extremely tender over infected tissue with hypoaesthesia to light touch
  - skin necrosis and crepitus/gas gangrene are late signs
- fever and tachycardia may be absent or occur late in the presentation

Management

- urgent surgical referral debridement
- intravenous antibiotics

Prognosis

- average mortality of 20%



Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

5 6

Necrotising fasciitis

Suggest link

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Score: **16.7%**

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A 33-year-old woman is brought to the emergency department by ambulance. She was found by her sister at home, confused and complaining of headache and fever. She has no past medical history and is on no regular medications. She had returned seven days previously from a trip on the Trans-Siberian Railway where she had stopped at a number of locations to do some hiking with friends. She had a brief flu-like illness towards the end of the holiday, which had resolved.

Her observations are heart rate 93 beats per minute, blood pressure 128/84 mmHg, respiratory rate 19/minute, oxygen saturations 96% on room air and temperature 38.4°C.

On examination, her Glasgow coma scale is 12/15 (eyes 3 verbal 4 motor 5). There is evidence of nuchal rigidity and when she opens her eyes she is photophobic. Examination is otherwise unremarkable and there is no evidence of rash.

Urinalysis is normal.

An ECG demonstrates sinus rhythm.

Plain radiography of the chest demonstrates clear lung fields.

Blood tests:

Hb	137 g/L	Male: (135-180) Female: (115 - 160)
Platelets	149 * 10^9/L	(150 - 400)
WBC	11.8 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	6.4 mmol/L	(2.0 - 7.0)
Creatinine	101 µmol/L	(55 - 120)
Glucose	4 mmol/L	(4-11)
CRP	38 mg/L	(< 5)

She is treated empirically with ceftriaxone and aciclovir for presumed meningo-encephalitis and a lumbar puncture is performed.

Results:

Appearance	clear	

WCC	80 cells/μL	(<5)
Cell type	lymphocytes	92%
Glucose	3.6 mmol/L	(2.8-4.2)
Protein	.65 g/L	(0.15 -0.45)

Subsequent testing reveals specific IgM antibodies in the CSF against a flavivirus.

What is the appropriate next step in management?

- ○ Continue aciclovir and ceftriaxone ×
- ○ Continue aciclovir and stop ceftriaxone ×
- ○ Continue ceftriaxone and stop aciclovir ×
- ○ Stop ceftriaxone and aciclovir and manage supportively ×
- ○ Stop ceftriaxone and aciclovir and start ganciclovir ×

Submit answer

Reference ranges

Score: **0%**

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Subsequent testing reveals specific IgM antibodies in the CSF against a flavivirus.

What is the appropriate next step in management?

- Continue aciclovir and ceftriaxone 6%
- Continue aciclovir and stop ceftriaxone 18%
- Continue ceftriaxone and stop aciclovir 2%
- Stop ceftriaxone and aciclovir and manage supportively 60%
- Stop ceftriaxone and aciclovir and start ganciclovir 14%

Treatment for tick-borne encephalitis is supportive

Important for me Less important

Stop ceftriaxone and aciclovir and manage supportively is the correct answer. The patient has tick-borne encephalitis. This is evidenced by the fact that she has returned from a region where the disease is endemic (Siberia) and has presented with a biphasic illness characterized by flu-like symptoms in the first stage and neurological dysfunction in the form of meningoencephalitis in the second stage. Her lumbar puncture results suggest a viral meningitis and her CSF testing reveal specific antibodies against a flavivirus, which is the cause of tick-borne encephalitis. There is no specific antiviral treatment for tick-borne encephalitis. Patients need admission to hospital if neurological symptoms are present and require supportive care in the form of analgesia, anti-pyretics, fluid and electrolyte management and intensive care input if appropriate.

Continue aciclovir and ceftriaxone is an incorrect answer. We have confirmed a diagnosis of tick-borne encephalitis. Ceftriaxone can be stopped as the CSF results do not indicate a bacterial infection and aciclovir can be stopped as we have a confirmed diagnosis that does not benefit from specific anti-viral treatment. Aciclovir is indicated in cases of meningo-encephalitis caused by herpes zoster and herpes simplex.

Continue aciclovir and stop ceftriaxone is incorrect. Aciclovir has no benefit in the management of tick-borne encephalitis.

Continue ceftriaxone and stop aciclovir is incorrect. The CSF results demonstrate a lymphocytic pleocytosis with a normal glucose, indicating a viral meningitis. Ceftriaxone has no role in the

management of viral infections.

Stop ceftriaxone and aciclovir and start ganciclovir is incorrect. Ganciclovir is predominantly used in the treatment of severe cytomegalovirus infection in immunocompromised patients. It has no role in the treatment of tick-borne encephalitis.

Discuss (2) | Improve

Next question >

Tick-borne encephalitis ★

Carried by ticks hosted by native wildlife, tick-borne encephalitis is actually caused by a virus (Flavivirus) and transmitted by the bite of an infected tick.

Once transmitted to the host, flavivirus infection manifests as a biphasic illness. During the first phase, patients may describe constitutional upset reporting headaches, myalgia, and fevers. There is typically then an asymptomatic period before the disease course progresses to phase two, which is characterized by infection by symptoms of central nervous system involvement, typically meningitis or, as the name suggests, encephalitis. The time period between phase one and two may be highly variable or absent in the case of severe infection.

Symptoms usually manifest within 7 days of infection, however, the incubation period can be up to a month. Long term neurological sequelae may persist for months - years following infection.

There are three species of flavivirus implicated in tick-borne encephalitis: European, Far Eastern and Siberian, with the Far Eastern species typically causing the most severe illness, often progressing rapidly to central nervous system involvement with no asymptomatic period, (European centre for Disease Prevention and Control quotes a mortality rate of around 35% in cases of Far Eastern flavivirus species infection).

Diagnosis is made on the basis of clinical suspicion, with patients showing characteristic symptoms and travel history confirming recent travel to affected areas. Diagnosis may be confirmed by cerebrospinal fluid (CSF) analysis demonstrating specific IgM or IgG antibodies.

Treatment is supportive, however, the addition of doxycycline or a cephalosporin may be advised if Lyme disease is considered a differential diagnosis until confirmation via CSF sampling can be obtained.

A vaccination available and recommended to those travelling to endemic areas and planning to engage in high-risk outdoor activities, for example, hiking in rural forested areas and/or grasslands. Precautions to avoid tick bites are recommended to all travellers to endemic areas.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 65-year-old man presents to the emergency department with three days of fever, leg pain and rash. He has a past medical history of type 2 diabetes, peripheral vascular disease and hypertension. He is on regular clopidogrel, amlodipine, metformin, gliclazide and insulin. He is independent and a retired shoe-maker.

His observations are, heart rate 121 beats per minute, blood pressure 84/56 mmHg, respiratory rate 24/minute, oxygen saturation 95% on room air and temperature 39.1°C.

On examination, he appears diaphoretic and unwell. His capillary refill time is four seconds and he is cool to touch. His pulse is regular and thready. Chest auscultation is normal. His abdomen is soft and non-tender. Heart sounds are normal and there are no murmurs. His left thigh is erythematous and painful to touch. The pain appears out of proportion to the clinical signs and the leg feels wooden and hard.

Blood tests:

Hb	108 g/L	Male: (135-180) Female: (115 - 160)
Platelets	555 * 10^9/L	(150 - 400)
WBC	21.1 * 10^9/L	(4.0 - 11.0)
Na^+	131 mmol/L	(135 - 145)
K^+	4.8 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	181 µmol/L	(55 - 120)
CRP	321 mg/L	(< 5)

What is the appropriate initial pharmacological management?

- Benzyl-penicillin and Flucloxacillin
- Flucloxacillin
- Intravenous immunoglobulin
- Tazocin and Clindamycin
- Vancomycin and Metronidazole

Submit answer

Reference ranges

Score: **0%**

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A 65-year-old man presents to the emergency department with three days of fever, leg pain and rash. He has a past medical history of type 2 diabetes, peripheral vascular disease and hypertension. He is on regular clopidogrel, amlodipine, metformin, gliclazide and insulin. He is independent and a retired shoe-maker.

His observations are, heart rate 121 beats per minute, blood pressure 84/56 mmHg, respiratory rate 24/minute, oxygen saturation 95% on room air and temperature 39.1°C.

On examination, he appears diaphoretic and unwell. His capillary refill time is four seconds and he is cool to touch. His pulse is regular and thready. Chest auscultation is normal. His abdomen is soft and non-tender. Heart sounds are normal and there are no murmurs. His left thigh is erythematous and painful to touch. The pain appears out of proportion to the clinical signs and the leg feels wooden and hard.

Blood tests:

Hb	108 g/L	Male: (135-180) Female: (115 - 160)
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Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	181 µmol/L	(55 - 120)
CRP	321 mg/L	(< 5)

What is the appropriate initial pharmacological management?

Benzyl-penicillin and Flucloxacillin	16%
Flucloxacillin	20%
Intravenous immunoglobulin	2%
Tazocin and Clindamycin	46%
Vancomycin and Metronidazole	17%

Type 1, or polymicrobial, necrotising fasciitis is the most common

Important for me Less important

Tazocin and clindamycin is the correct answer. This man has necrotizing fasciitis as evidenced by an erythematous rash with pain out of proportion to the clinical signs, fever, a tense thigh and shock with organ dysfunction (acute kidney injury). The most common type of necrotizing fasciitis is type 1, which is polymicrobial. We, therefore, need an antibiotic combination that provides adequate coverage against gram-positive, gram-negative and anaerobic bacteria. Tazocin and clindamycin is the only option listed that fulfils these criteria. Clindamycin has the additional benefit of theoretically inhibiting toxin production in severe streptococcal infections. The polymicrobial type is in particular, more common in diabetic patients.

Benzyl-penicillin and Flucloxacillin are not correct. This will only provide adequate coverage for streptococcal and staphylococcal skin and soft tissue infections (gram-positive) but will not adequately treat gram-negative and anaerobic infections.

Flucloxacillin is incorrect. Again, this has activity predominantly against gram-positive bacteria.

Intravenous immunoglobulin is incorrect. This treatment can be used as an adjunct in severe skin and soft tissue infections but it does not replace antibiotic treatment.

Vancomycin and metronidazole are incorrect. Vancomycin provides coverage for gram-positive organisms and metronidazole for anaerobic bacteria but there is not sufficient gram-negative coverage in this combination.

Discuss (3) Improve

Next question

Necrotising fasciitis

Necrotising fasciitis is a medical emergency that is difficult to recognise in the early stages.

It can be classified according to the causative organism:

- type 1 is caused by mixed anaerobes and aerobes (often occurs post-surgery in diabetics). This is the most common type
- type 2 is caused by *Streptococcus pyogenes*

Risk factors

- skin factors: recent trauma, burns or soft tissue infections
- diabetes mellitus

    - the most common preexisting medical condition
    - particularly if the patient is treated with SGLT-2 inhibitors
- intravenous drug use
- immunosuppression

The most commonly affected site is the perineum (Fournier's gangrene).

Features
- acute onset
- pain, swelling, erythema at the affected site
    - often presents as rapidly worsening cellulitis with pain out of keeping with physical features
    - extremely tender over infected tissue with hypoaesthesia to light touch
    - skin necrosis and crepitus/gas gangrene are late signs
- fever and tachycardia may be absent or occur late in the presentation

Management
- urgent surgical referral debridement
- intravenous antibiotics

Prognosis
- average mortality of 20%



Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

5 6

Necrotising fasciitis

Suggest link Report broken link

Score: **16.7%**

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A 32-year-old man presents with his left eyelid closing and dribbling from the side of his mouth. This arose three weeks ago and he has also felt tired and fatigued. He denies any fevers and has no weakness in any other parts of his body. He has a dry cough which comes and goes and he put down to hay fever. His left ear has reduced hearing and has been sore.

In addition, there is a red rash on his legs which has now faded. He has no past medical history, has not travelled abroad and is regularly sexually active with a single male partner. He has had shingles three months ago.

On examination, he has a clear left sided facial droop with normal sensation and no sparing of the forehead. He has normal power in all four limbs with no sensory loss. There is a faint red circle on the right leg surrounding a central red area within. Scattered crepitations are present in the lungs. He has pain in his left ear with an effusion behind the tympanic membrane.

Hb	156 g/l	Na^+	135 mmol/l
Platelets	345 * 10^9/l	K^+	4.5 mmol/l
WBC	12 * 10^9/l	Urea	4.2 mmol/l
Neuts	9 * 10^9/l	Creatinine	94 µmol/l
Lymphs	1 * 10^9/l	CRP	34 mg/l
Eosin	0.2 * 10^9/l		
Corrected Calcium	2.2mmol/l		

ECG	sinus, PR interval 202ms
Chest x-ray	no abnormalities seen

What is the likely diagnosis?

- Ramsay hunt syndrome
- Lyme disease
- Sarcoidosis
- Bell's palsy
- Syphilis

Submit answer

Reference ranges

Score: **0%**

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Eosin	0.2 * 10^9/l		
Corrected Calcium	2.2mmol/l		

ECG	sinus, PR interval 202ms
Chest x-ray	no abnormalities seen

What is the likely diagnosis?

Ramsay hunt syndrome	19%
Lyme disease	72%
Sarcoidosis	2%
Bell's palsy	2%
Syphilis	5%

Lyme's disease can present with a facial nerve palsy

Important for me Less important

This gentleman has a facial nerve palsy without sparing of the forehead. Ramsay hunt syndrome occurs following shingles in the facial nerve and has vesicles in the ear canal but this is visible and it does not cause an effusion. Sarcoidosis is possible but the calcium is normal and there is no hilar lymphadenopathy. Bell's palsy is idiopathic or even possibly viral and should be diagnosed once all other causes are excluded. Syphilis is possible and can have a rash however the rash here is described as a red circle with a central part which should prompt thoughts of erythema migrans in Lyme disease.

Discuss (6) Improve

Next question >

Lyme disease

Lyme disease is caused by the spirochaete *Borrelia burgdorferi* and is spread by ticks.

Early features (within 30 days)

- erythema migrans
  - 'bulls-eye' rash is typically at the site of the tick bite
  - typically develops 1-4 weeks after the initial bite but may present sooner
  - usually painless, more than 5 cm in diameter and slowlly increases in size
  - present in around 80% of patients.
- systemic features
  - headache
  - lethargy
  - fever
  - arthralgia





Later features (after 30 days)

- cardiovascular
  - heart block
  - peri/myocarditis

- neurological
  - facial nerve palsy
  - radicular pain
  - meningitis



Investigation

- NICE recommend that Lyme disease can be diagnosed clinically if erythema migrans is present
  - erythema migrans is therefore an indication to start antibiotics
- enzyme-linked immunosorbent assay (ELISA) antibodies to *Borrelia burgdorferi* are the first-line test
  - if negative and Lyme disease is still suspected in people tested within 4 weeks from symptom onset, repeat the ELISA 4-6 weeks after the first ELISA test. If still suspected in people who have had symptoms for 12 weeks or more then an immunoblot test should be done
  - if positive or equivocal then an immunoblot test for Lyme disease should be done

Management of asymptomatic tick bites

- tick bites can be a relatively common presentation to GP practices, and can cause significant anxiety
- if the tick is still present, the best way to remove it is using fine-tipped tweezers, grasping the tick as close to the skin as possible and pulling upwards firmly. The area should be washed following.
- NICE guidance does not recommend routine antibiotic treatment to patients who've suffered a tick bite

Management of suspected/confirmed Lyme disease

- doxycycline if early disease. Amoxicillin is an alternative if doxycycline is contraindicated (e.g. pregnancy)
  - people with erythema migrans should be commenced on antibiotic without the need for further tests
- ceftriaxone if disseminated disease
- Jarisch-Herxheimer reaction is sometimes seen after initiating therapy: fever, rash, tachycardia after first dose of antibiotic (more commonly seen in syphilis, another spirochaetal disease)

AI + Q 123

Next question >

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Links

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Lyme disease: management

NICE

11 10

2018 Lyme disease guidelines

Dermnet NZ

12 5

Lyme disease

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Media



Lyme disease

Osmosis - YouTube 4 1

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You are working in the liver clinic. A 28-year-old pregnant lady attends the clinic. She has been diagnosed with hepatitis C. She has no other co-morbidities and is not taking any regular medications. Hepatitis B and HIV have been excluded. She asks you about the risk to her baby.

What is the risk of vertical transmission?

- 30%
- 6%
- 80%
- 50%
- No risk

Submit answer

Reference ranges

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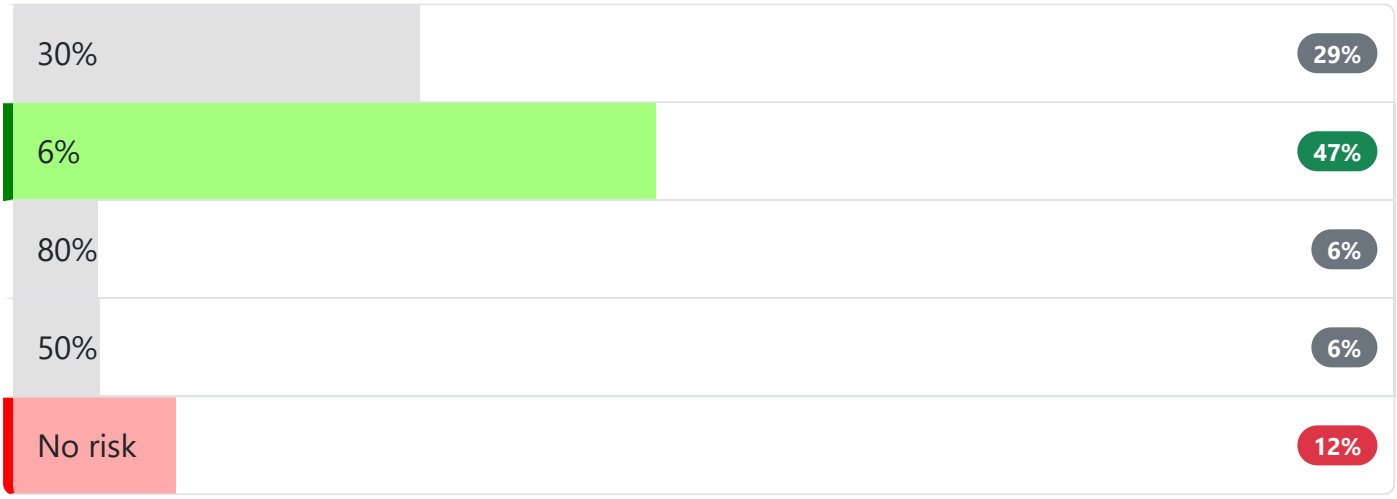
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What is the risk of vertical transmission?



Risk of vertical transmission for hepatitis C is 6%

Important for me Less important

Risk of vertical transmission with hepatitis C is generally low and up to 6%. The risk of transmission appears to be high in women with HIV co-infection or high viral load.

Discuss (1) Improve

Next question >

Hepatitis C

Hepatitis C is likely to become a significant public health problem in the UK in the next decade. It is thought around 200,000 people are chronically infected with the virus. At risk groups include intravenous drug users and patients who received a blood transfusion prior to 1991 (e.g. haemophiliacs).

Pathophysiology

- hepatitis C is a RNA flavivirus
- incubation period: 6-9 weeks

Transmission

- the risk of transmission during a needle stick injury is about 2%
- the vertical transmission rate from mother to child is about 6%. The risk is higher if there is coexistent HIV
- breastfeeding is not contraindicated in mothers with hepatitis C
- the risk of transmitting the virus during sexual intercourse is probably less than 5%
- there is no vaccine for hepatitis C

After exposure to the hepatitis C virus only around 30% of patients will develop features such as:

- a transient rise in serum aminotransferases / jaundice
- fatigue
- arthralgia

Investigations

- HCV RNA is the investigation of choice to diagnose acute infection
- whilst patients will eventually develop anti-HCV antibodies it should be remembered that patients who spontaneously clear the virus will continue to have anti-HCV antibodies

Outcome

- around 15-45% of patients will clear the virus after an acute infection (depending on their age and underlying health) and hence the majority (55-85%) will develop chronic hepatitis C

Chronic hepatitis C

Chronic hepatitis C may be defined as the persistence of HCV RNA in the blood for 6 months.

Potential complications of chronic hepatitis C

- rheumatological problems: arthralgia, arthritis
- eye problems: Sjogren's syndrome
- cirrhosis (5-20% of those with chronic disease)
- hepatocellular cancer
- cryoglobulinaemia: typically type II (mixed monoclonal and polyclonal)
- porphyria cutanea tarda (PCT): it is increasingly recognised that PCT may develop in patients with hepatitis C, especially if there are other factors such as alcohol abuse
- membranoproliferative glomerulonephritis

Management of chronic infection

- treatment depends on the viral genotype - this should be tested prior to treatment
- the management of hepatitis C has advanced rapidly in recent years resulting in clearance rates of around 95%. Interferon based treatments are no longer recommended

- the aim of treatment is sustained virological response (SVR), defined as undetectable serum HCV RNA six months after the end of therapy
- currently a combination of protease inhibitors (e.g. daclatasvir + sofosbuvir or sofosbuvir + simeprevir) with or without ribavirin are used

Complications of treatment

- ribavirin - side-effects: haemolytic anaemia, cough. Women should not become pregnant within 6 months of stopping ribavirin as it is teratogenic
- interferon alpha - side-effects: flu-like symptoms, depression, fatigue, leukopenia, thrombocytopenia



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 7 5

Primary care management of people with confirmed active hepatitis C

European Association for the Study of the Liver 13 2

2018 EASL Recommendations on Treatment of Hepatitis C

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Media



Viral hepatitis

Osmosis - YouTube 1 0



Hepatitis

Podmedics - YouTube 0 0



Anti-hepatitis medications

Osmosis - YouTube 0 0

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A 55-year-old patient is admitted to the acute medical unit with a 10-day history of a dry cough and progressive shortness of breath, particularly on exertion. He also complains of intermittent fevers and a general loss of appetite. His past medical history includes HIV and he is non-compliant with his regular medications.

His observations are as follows:

- Temperature 38.3ºC
- Heart rate 112 beats/min
- Blood pressure 123/84mmHg
- Respiratory rate 25 breaths/min
- Oxygen saturations 94% on air, 83% on exertion

On examination, he appears pale and cachectic. There are audible crackles bilaterally. His heart rate is regular and heart sounds are normal. He has a soft and non-tender abdomen.

Laboratory tests:

CD4 count	< 200/mm³

Which of the following complications is most commonly associated with the likely underlying diagnosis?

- Encephalitis
- Hepatitis
- Pancreatitis
- Pleural effusion
- Pneumothorax

Submit answer

Reference ranges

Score: **0%**

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On examination, he appears pale and cachectic. There are audible crackles bilaterally. His heart rate is regular and heart sounds are normal. He has a soft and non-tender abdomen.

Laboratory tests:

CD4 count	< 200/mm³

Which of the following complications is most commonly associated with the likely underlying diagnosis?

Encephalitis	7%
Hepatitis	2%
Pancreatitis	3%
Pleural effusion	14%
Pneumothorax	**74%**

Pneumocystis jiroveci pneumonia - pneumothorax is a common complication

Important for me Less important

This patient has a diagnosis of pneumocystis jirovecii pneumonia (or PCP), a fungal pneumonia that is most commonly seen in immunocompromised individuals, particularly those with HIV. Symptoms of infection include fever, dry cough and shortness of breath (especially on exertion) as

seen in this patient. Given his lack of compliance with his medications, most importantly the highly active antiretroviral therapy, combined with the CD4 count < 200/mm^3, this patient is at high risk of developing PCP. The most common complication associated with PCP is a pneumothorax secondary to rupture of pulmonary cysts.

Extrapulmonary manifestations of PCP include hepatosplenomegaly and lymphadenopathy. However, these are rare and less likely. Encephalitis is a complication of several viral infections including measles, herpes simplex and varicella-zoster virus. It is not a complication of PCP.

Hepatitis has multiple causes such as inflammatory, ischaemic and infective (e.g. hepatitis A,B,C,D and E). PCP is not a cause of hepatitis.

Pancreatitis is a complication of mumps and coxsackie B virus, but not PCP.

Along with pneumothorax, other respiratory complications of PCP include acute respiratory distress syndrome. However, a pleural effusion is a less likely complication compared to a pneumothorax, making it incorrect.

Discuss (2) | Improve

Next question >

HIV: *Pneumocystis jiroveci* pneumonia

Whilst the organism *Pneumocystis carinii* is now referred to as *Pneumocystis jiroveci*, the term *Pneumocystis carinii* pneumonia (PCP) is still in common use

- *Pneumocystis jiroveci* is an unicellular eukaryote, generally classified as a fungus but some authorities consider it a protozoa
- PCP is the most common opportunistic infection in AIDS
- all patients with a CD4 count < 200/mm^3 should receive PCP prophylaxis

Features

- dyspnoea
- dry cough
- fever
- very few chest signs

Pneumothorax is a common complication of PCP.

Extrapulmonary manifestations are rare (1-2% of cases), may cause

- hepatosplenomegaly
- lymphadenopathy

- choroid lesions

Investigation

- CXR: typically shows bilateral interstitial pulmonary infiltrates but can present with other x-ray findings e.g. lobar consolidation. May be normal
- exercise-induced desaturation
- sputum often fails to show PCP, bronchoalveolar lavage (BAL) often needed to demonstrate PCP (silver stain shows characteristic cysts)





Management

- co-trimoxazole
- IV pentamidine in severe cases
- aerosolized pentamidine is an alternative treatment for *Pneumocystis jiroveci* pneumonia but is less effective with a risk of pneumothorax
- steroids if hypoxic (if pO2 < 9.3kPa then steroids reduce risk of respiratory failure by 50% and death by a third)

AI + Q 123

Next question ›

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Textbooks

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Links

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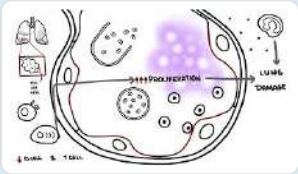
Pneumocystis pneumonia

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Pneumocystis Jirovecii Pneumonia review

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Media



Pneumocystis jirovecii - Pneumocystis pneumonia

Armando Hasudungan - YouTube 0 0



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube 2 1

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A 28-year-old man presents with cough and fevers. He states that the cough has been going on for approximately 6 months. He states that there is occasional blood in the sputum. He has also lost approximately 10 kg over the past 2 months. He immigrated to the UK from India approximately 1 year ago. He is not aware of being in contact with anyone with tuberculosis.

You suspect a diagnosis of tuberculosis and isolate him into a negative pressure room.

What investigation is most likely to yield a positive diagnosis?

- Bronchoalveolar lavage (BAL) with staining, culture and sensitivity
- Induced sputum with staining, culture and sensitivity
- Interferon-Gamma Release Assays
- Mantoux test
- Chest x-ray

Submit answer

Reference ranges

Score: **0%**

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You suspect a diagnosis of tuberculosis and isolate him into a negative pressure room.

What investigation is most likely to yield a positive diagnosis?

Bronchoalveolar lavage (BAL) with staining, culture and sensitivity	48%
Induced sputum with staining, culture and sensitivity	30%
Interferon-Gamma Release Assays	17%
Mantoux test	3%
Chest x-ray	2%

In order to confirm the diagnosis of TB, it is necessary to culture *Mycobacterium tuberculosis* from a respiratory specimen. Bronchoscopy will produce the best respiratory specimen for acid-fast staining and mycobacterial culture

Important for me Less important

Patients who are suspected to have pulmonary tuberculosis should have multiple respiratory samples (3 deep cough sputum samples, preferably including 1 early morning sample) for TB microscopy and culture. Patients that are unable to produce sputum spontaneously or have smear-negative sputum present a diagnostic challenge. NICE recommend that clinicians should perform induction of sputum or bronchoscopy and lavage in adults in this situation.

The investigation with the greatest diagnostic yield is bronchoscopy with lavage making this the best answer.

The Mantoux and Interferon-Gamma Release Assays tests are useful for diagnosing latent TB infection.

Next question

Tuberculosis: types

Tuberculosis (TB) is an infection caused by *Mycobacterium tuberculosis* that most commonly affects the lungs. Understanding the pathophysiology of TB can be difficult - the key is to differentiate between primary and secondary disease.

Primary tuberculosis

A non-immune host who is exposed to *M. tuberculosis* may develop a primary infection of the lungs. A small lung lesion known as a Ghon focus develops. The Ghon focus is composed of tubercle-laden macrophages. The combination of a Ghon focus and hilar lymph nodes is known as a Ghon complex

In immunocompetent people, the initial lesion usually heals by fibrosis. Those who are immunocompromised may develop disseminated disease (miliary tuberculosis).

Secondary (post-primary) tuberculosis

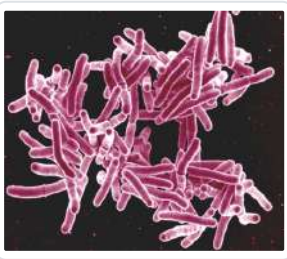
If the host becomes immunocompromised the initial infection may become reactivated. Reactivation generally occurs in the apex of the lungs and may spread locally or to more distant sites. Possible causes of immunocompromise include:

- immunosuppressive drugs including steroids
- HIV
- malnutrition

The lungs remain the most common site for secondary tuberculosis. Extra-pulmonary infection may occur in the following areas:

- central nervous system (tuberculous meningitis - the most serious complication)
- vertebral bodies (Pott's disease)
- cervical lymph nodes (scrofuloderma)
- renal
- gastrointestinal tract





Next question

Textbooks

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Links

NICE

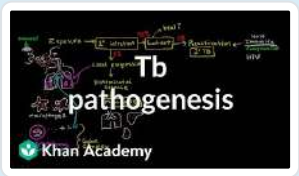
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2016 Tuberculosis guidelines

Suggest link

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Media



TB pathogenesis

Khan Academy - YouTube 1 0



Tuberculosis

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A 38 year-old intravenous drug user presented to accident and emergency with a 10-day history of severe watery diarrhoea and abdominal cramps. Her past medical history included treatment for a groin abscess 3 years previously. She could not remember ingesting any possible causative food and had not travelled abroad. She smoked 15 cigarettes per day and drank 30-40 units of alcohol per week.

On examination, her temperature was 36.8ºC, heart rate 100 beats per minute, blood pressure 92/58 mmHg, respiratory rate 18 breaths per minute and oxygen saturations 100% on room air. Her tongue appeared dry with white patches evident on the hard palate and throat. The JVP was not visible. Her chest was clear on auscultation and heart sounds were normal. The abdomen was soft on palpation with some mild central abdominal tenderness.

Investigations:

Haemoglobin	170 g/L
White cell count	4.5 * 10^9/l
Neutrophil count	3.0 * 10^9/l
Lymphocyte count	0.1 * 10^9/l
Eosinophil count	0.4 * 10^9/l
Platelets	423 * 10^9/l

Sodium	132 mmol/l
Potassium	3.0 mmol/L
Urea	13.0 mmol/L
Creatinine	110 mol/L
Alkaline phosphatase	57 IU/L
Alanine aminotransferase	60 IU/L
Gamma-glutyl transferase	67 IU/L
Bilirubin	19 mol/L
Albumin	29 g/L

Abdominal x-ray: No abnormality detected
Stool culture: No growth at 48 hours

What additional staining technique should be used for analysing the stool sample?

- Ziehl-Neelson
- Romanowsky
- Periodic acid-Schiff
- Sudan
- Silver

Submit answer

Reference ranges

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Bilirubin	19 mol/L
Albumin	29 g/L

Abdominal x-ray: No abnormality detected
Stool culture: No growth at 48 hours

What additional staining technique should be used for analysing the stool sample?

Ziehl-Neelson	50%
Romanowsky	5%
Periodic acid-Schiff	19%
Sudan	7%
Silver	20%

This patient is likely to have *Cryptosporidium parvum* infection. In the immunocompetent, the protozoan causes a self-limiting diarrhoeal illness lasting 7-10 days or may even be asymptomatic. However, in this case there is likely to be co-existing immunosuppression from human immunodeficiency virus infection, which can result in a severe cholera-like diarrhoea syndrome. The most appropriate staining technique in this case is a modified acid-fast stain such as Ziehl-Neelson, which will stain *C. parvum* oocysts red. Periodic acid-schiff is used on jejunal biopsy specimens to diagnose Whipples disease. Silver staining can pick up *Pneumocystis jiroveci* on sputum samples. Sudan stain is used to diagnose steatorrhoea, and Romanowsky is a blood staining method which preceded Giemsa staining.

Discuss (4) Improve

Next question

HIV: diarrhoea

Diarrhoea is common in patients with HIV. This may be due to the effects of the virus itself (HIV enteritis) or opportunistic infections

Possible causes

- Cryptosporidium + other protozoa (most common)
- Cytomegalovirus
- *Mycobacterium avium intracellulare*
- Giardia

Cryptosporidium is the most common infective cause of diarrhoea in HIV patients. It is an intracellular protozoa and has an incubation period of 7 days. Presentation is very variable, ranging from mild to severe diarrhoea. A modified Ziehl-Neelsen stain (acid-fast stain) of the stool may reveal the characteristic red cysts of Cryptosporidium. Treatment is difficult, with the mainstay of management being supportive therapy*

Mycobacterium avium intracellulare is an atypical mycobacteria seen with the CD4 count is below 50. Typical features include fever, sweats, abdominal pain and diarrhoea. There may be hepatomegaly and deranged LFTs. Diagnosis is made by blood cultures and bone marrow examination. Management is with rifabutin, ethambutol and clarithromycin

*nitazoxanide is licensed in the US for immunocompetent patients

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 23-year-old man attends the emergency department after arriving back in the UK from a one-year trip travelling in South America. He denies any acute medical problems but reports that he is concerned about a dog bite he had received in Brazil about three months previously. The patient had been on a rain-forest expedition when a dog from the village he was staying in became aggressive and bit him on the left hand, leaving a open wound. The local inhabitants of the village had then chased the dog away. The patient had received first aid from his tour guide and been provided with a course of oral antibiotics. The wound had then healed over the following two weeks. It was only when the patient had been speaking with fellow travellers shortly before he returned to the UK did he realise the possible risk for rabies exposure.

The patient was well in himself and denied any neurological or other symptoms. He had no significant past medical history and took no regular medications. He admitted that he had no adequately researched his travel health needs before leaving the UK and so had not had any vaccinations prior to his trip.

Examination of the patients wound demonstrated fully healed scars consistent with a bite marks across the palm and dorsal surface of the wrist. The limb had normal neurological and vascular examination and the patient reported normal function of the hand.

What is the correct statement regarding rabies post-exposure prophylaxis for this patient?

- The patient should receive rabies vaccination but not rabies immunoglobulin as his wound has now healed
- The patient does not require post-exposure prophylaxis as he remains free of symptoms following the incubation period of rabies
- The patient should receive rabies immunoglobulin and full rabies vaccination schedule
- The patient does not require rabies post-exposure prophylaxis as he did not receive his injury in a high-risk region
- The patient should receive rabies immunoglobulin but not rabies vaccination as this is only of benefit if given pre-exposure

Submit answer

Reference ranges

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What is the correct statement regarding rabies post-exposure prophylaxis for this patient?

The patient should receive rabies vaccination but not rabies immunoglobulin as his wound has now healed	24%
The patient does not require post-exposure prophylaxis as he remains free of symptoms following the incubation period of rabies	18%
The patient should receive rabies immunoglobulin and full rabies vaccination schedule	50%
The patient does not require rabies post-exposure prophylaxis as he did not receive his injury in a high-risk region	1%
The patient should receive rabies immunoglobulin but not rabies vaccination as this is only of benefit if given pre-exposure	6%

The patient requires immediate (if belated) rabies post-exposure prophylaxis. Penetrating wounds are considered high risk-exposures, especially those to highly innervated areas such as the hands. The history of an unprovoked animal biting is also of concern.

Rabies immunoglobulin should be given no matter how long has elapsed since the exposure. As

much as possible of the immunoglobulin dose should be infiltrated around the wound, with the remainder given as an IM gluteal injection. This should be combined with a full course of rabies vaccination (typically a 5-dose schedule with doses at day 0, 3, 7, 14 and 28). Such post-exposure prophylaxis is highly effective in preventing the rabies virus reaching the nervous system.

Large parts of South America and Central America (including Brazil) are considered high-risk rabies regions. The virus is also endemic in Africa, the Middle East, Eastern Europe and most of Asia.

The incubation period of rabies can be prolonged and the patient is certainly still at risk of contracting the disease. One study found a mean incubation time of 274 days (range 12 days to 10 years).

Crowcroft N, Thampi N. The prevention and management of rabies. BMJ 2015;350:g7827.

http://www.who.int/rabies/human/adminimmuno/en/

Discuss (5) | Improve

Next question >

Rabies

Rabies is a viral disease that causes an acute encephalitis. The rabies virus is classed as a RNA rhabdovirus (specifically a lyssavirus) and has a bullet-shaped capsid. The vast majority of cases are caused by dog bites but it may also be transmitted by bat, raccoon and skunk bites.

After entry, the virus replicates in muscle tissue near the site of the bite before entering the peripheral nervous system. It then travels up to the central nervous system, where it causes encephalitis, leading to severe neurological symptoms. The virus eventually reaches the salivary glands, facilitating transmission.

Rabies is estimated to still kill around 25,000-50,000 people across the world each year. The vast majority of the disease burden falls on people in poor rural areas of Africa and Asia. Children are particularly at risk.

Features

- prodrome: headache, fever, agitation
- hydrophobia: water-provoking muscle spasms
- hypersalivation
- Negri bodies: cytoplasmic inclusion bodies found in infected neurons

There is now considered to be 'no risk' of developing rabies following an animal bite in the UK and the majority of developed countries. Following an animal bite in at-risk countries:

- the wound should be washed
- if an individual is already immunised then 2 further doses of vaccine should be given
- if not previously immunised then human rabies immunoglobulin (HRIG) should be given along with a full course of vaccination. If possible, the dose should be administered locally around the wound

If untreated the disease is nearly always fatal.

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Next question >

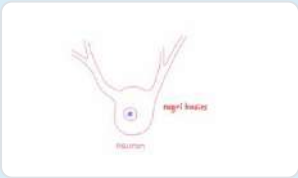
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Textbooks

High-yield textbook

Extended textbook

Media



Rabies

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A 35-year-old woman attends gastroenterology clinic following a referral from her general practitioner. The patient is suffering from ongoing gastrointestinal symptoms following a recent diagnosis of giardiasis, despite receiving appropriate first line treatment. The patient reports a 3-month history of variable bowel habit, consisting of periods of normal motions interspersed with episodes of frequent diarrhoea. During the course of her illness, the patient reports having lost approximately 7 kg in weight. The patient denied every experiencing fever or bloody diarrhoea, but did report unusual episodes of burping with an unpleasant taste, described by the patient as tasting like 'rotten eggs'.

After her initial symptoms, the patient attended her GP, with a subsequent stool microscopy test demonstrating evidence of infection with *Giardia lamblia*. The patient had not undertaken any recent overseas travel but was employed as a nursery nurse, which was suspected of being the source of her infection. Her employer had moved the patient to a different job role, not including direct contact with children after the patient disclosed her condition.

After the diagnosis of giardiasis, the patient was treated with a 5-day course of metronidazole consistent with British National Formulary guidelines. Following treatment, the patient reported some improvement but had not gained full resolution of her symptoms.

The patient was normally fit and well and her only significant past medical history was a salpingectomy performed 5 years previously due to an ectopic pregnancy. She had never previously experienced problematic gastrointestinal symptoms. The patient took no regular medications and had no known drug allergies.

Examination of the patient's abdomen was unremarkable and she appeared to be in generally good physical condition. A urinary pregnancy test was negative.

Following her review in the clinic, a decision was made to prescribe the patient a single dose of tinidazole as a second-line treatment for giardiasis. What advice should be given to the patient alongside this treatment?

- Avoid consuming legumes for 2-6 weeks
- Avoid consuming dairy products for 2-6 weeks
- Avoid consuming eggs for 2-6 weeks
- Her ongoing symptoms are likely secondary to irritable bowel syndrome not giardiasis
- Avoid consuming gluten for 2-6 weeks

Submit answer

Reference ranges

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Avoid consuming legumes for 2-6 weeks	16%
Avoid consuming dairy products for 2-6 weeks	53%
Avoid consuming eggs for 2-6 weeks	9%
Her ongoing symptoms are likely secondary to irritable bowel syndrome not giardiasis	10%
Avoid consuming gluten for 2-6 weeks	13%

The patient has typical symptoms of giardiasis. While giardiasis is the most common protozoal infection in returning travellers, research suggests that the majority of cases in the UK are acquired through domestic exposure such as child-care, sexual contact or consumption of unprocessed foods.

Following unsuccessful first line treatment, a repeat course of antibiotic therapy (either metronidazole or tinidazole) should be given. The patient has already taken steps to avoid reinfection by temporarily changing her job role away from direct contact with children. Giardiasis can cause temporary lactose intolerance, which can lead to ongoing gastrointestinal symptoms after the infection has been eradicated. To exclude this possibility, patients should be advised to avoid dairy products for 2-6 weeks.

There is an overlap between the symptoms of irritable bowel syndrome and giardiasis. Therefore, in an individual with symptoms suggestive of irritable bowel syndrome but with risk factors for *Giardia* infection, faecal testing for ova and parasites should be considered to exclude giardiasis. However, in this patient, the absence of gastrointestinal symptoms prior to the current episode would make a diagnosis of irritable bowel syndrome unlikely.

Minetti C, Chalmers R, Beeching N, Probert C, Lamden K. Giardiasis. BMJ 2016;355:i5369.

Discuss (7) | Improve

Next question >

Giardiasis ★

Giardiasis is caused by the flagellate protozoan *Giardia lamblia*. It is spread by the faeco-oral route.

Risk factors

- foreign travel
- swimming/drinking water from a river or lake
- male-male sexual contact

Features

- often asymptomatic
- non-bloody diarrhoea
  - steatorrhoea
- bloating, abdominal pain
- lethargy
- flatulence
- weight loss

- malabsorption and lactose intolerance can occur

Investigations

- stool microscopy for trophozoite and cysts: sensitivity of around 65%
- stool antigen detection assay: greater sensitivity and faster turn-around time than conventional stool microscopy methods
- PCR assays are also being developed

Treatment is with metronidazole.

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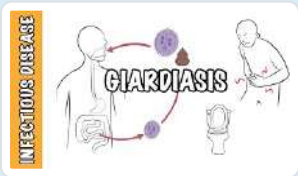
Next question

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Media



Giardiasis

Armando Hasudungan - YouTube 2 1

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A 31-year-old man presents to the emergency department with an episode of collapse. He reports that for the past week, he has been having fevers, headaches, and generally feels faint. He has recently returned from a two-month holiday to the North-Eastern coast of the United States of America (USA). He has no past medical history.

Blood results are as follows:

Hb	141 g/L	135-180
Platelets	231 * 10^9/L	(150 - 400)
WBC	19.3 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.1 mmol/L	(3.5 - 5.0)
Urea	5.1 mmol/L	(2.0 - 7.0)
Creatinine	91 µmol/L	(55 - 120)
CRP	74 mg/L	(< 5)



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Which infectious cause is most likely to be responsible?

- American trypanosomiasis
- Coxsackie A
- Coxsackie B
- Group A streptococcus
- Lyme disease

Submit answer

Reference ranges

Score: **0%**

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CRP	74 mg/L	(< 5)



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Which infectious cause is most likely to be responsible?

American trypanosomiasis	33%
Coxsackie A	4%
Coxsackie B	6%
Group A streptococcus	1%
Lyme disease	56%

This man's ECG shows a complete heart block with complete dissociation of the QRS complexes from the p waves. The most common infectious cause for this is Lyme disease which is the correct answer here. Ticks that transmit Lyme disease can be found in the United Kingdom as well as the USA.

American trypanosomiasis causes Chagas' disease which can manifest as myocarditis and, ultimately, arrhythmias including complete atrioventricular block. Despite the name, this disease is mainly found in Southern and Central America, whereas the main source of transmission throughout the USA is via immigration and blood transfusions.

Coxsackie A is not associated with ECG changes and therefore this answer is incorrect.

Coxsackie B can cause viral myocarditis for which the most common ECG change is sinus tachycardia. Fulminant Coxsackie myocarditis can result in complete heart block however this would likely present with gastrointestinal symptoms and more severe signs of cardiogenic shock.

Group A streptococci can cause rheumatic fever which in turn can cause first-degree heart block however complete heart block is a far rarer observation.

Discuss (3) | Improve

Next question >

Lyme disease ★

Lyme disease is caused by the spirochaete *Borrelia burgdorferi* and is spread by ticks.

Early features (within 30 days)

- erythema migrans
  - 'bulls-eye' rash is typically at the site of the tick bite
  - typically develops 1-4 weeks after the initial bite but may present sooner
  - usually painless, more than 5 cm in diameter and slowlly increases in size
  - present in around 80% of patients.
- systemic features
  - headache
  - lethargy
  - fever
  - arthralgia





Later features (after 30 days)

- cardiovascular
  - heart block
  - peri/myocarditis
- neurological
  - facial nerve palsy
  - radicular pain
  - meningitis



Investigation

- NICE recommend that Lyme disease can be diagnosed clinically if erythema migrans is present
  - erythema migrans is therefore an indication to start antibiotics
- enzyme-linked immunosorbent assay (ELISA) antibodies to *Borrelia burgdorferi* are the first-line test
  - if negative and Lyme disease is still suspected in people tested within 4 weeks from symptom onset, repeat the ELISA 4-6 weeks after the first ELISA test. If still suspected in people who have had symptoms for 12 weeks or more then an immunoblot test should be done
  - if positive or equivocal then an immunoblot test for Lyme disease should be done

Management of asymptomatic tick bites

- tick bites can be a relatively common presentation to GP practices, and can cause significant anxiety
- if the tick is still present, the best way to remove it is using fine-tipped tweezers, grasping the tick as close to the skin as possible and pulling upwards firmly. The area should be washed following.
- NICE guidance does not recommend routine antibiotic treatment to patients who've suffered a tick bite

Management of suspected/confirmed Lyme disease

- doxycycline if early disease. Amoxicillin is an alternative if doxycycline is contraindicated (e.g. pregnancy)

    - people with erythema migrans should be commenced on antibiotic without the need for further tests
- ceftriaxone if disseminated disease
- Jarisch-Herxheimer reaction is sometimes seen after initiating therapy: fever, rash, tachycardia after first dose of antibiotic (more commonly seen in syphilis, another spirochaetal disease)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 11 13

Lyme disease: management

NICE 11 10

2018 Lyme disease guidelines

Dermnet NZ 12 5

Lyme disease

Suggest link Report broken link

Media



Lyme disease

Osmosis - YouTube 4 1

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A 28 year-old man presents to his doctor with a rash and bone pain. Radiographs of the limbs revealed numerous osteolytic lesions. He was successfully treated for secondary syphilis.

Which of the following tests is likely to remain positive in this patient despite treatment?

- Blood culture
- Treponema pallidum particle agglutination (TPPA)
- Venereal disease reference laboratory (VDRL)
- Rapid plasmin reagin (RPR)
- Wasserman test

Submit answer

Reference ranges

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Back to top

A 28 year-old man presents to his doctor with a rash and bone pain. Radiographs of the limbs revealed numerous osteolytic lesions. He was successfully treated for secondary syphilis.

Which of the following tests is likely to remain positive in this patient despite treatment?

Blood culture	1%
Treponema pallidum particle agglutination (TPPA)	58%
Venereal disease reference laboratory (VDRL)	29%
Rapid plasmin reagin (RPR)	11%
Wasserman test	1%

Syphilis is a disease caused by infection with the spirochete Treponema pallidum. The infection is systemic and the disease is characterized by periods of latency. These features, together with the fact that Treponema pallidum cannot be isolated in culture, mean that serologic techniques play a major role in the diagnosis and follow-up of treatment for syphilis.

The Treponema pallidum particle agglutination assay (also called TPPA test) is an indirect agglutination assay used for detection and titration of antibodies against Treponema pallidum.

IgG antibodies to syphilis can remain elevated despite appropriate antimicrobial treatment and a reactive result does not distinguish between recent or past infection.

Discuss (7) | Improve

Next question >

Syphilis: investigation

Treponema pallidum is a very sensitive organism and cannot be grown on artificial media. The diagnosis is therefore usually based on clinical features, serology and microscopic examination of infected tissue.

Serological tests can be divided into:

- non-treponemal tests

    - not specific for syphilis, therefore may result in false positives (see below)
    - based upon the reactivity of serum from infected patients to a cardiolipin-cholesterol-lecithin antigen
    - assesses the quantity of antibodies being produced
    - becomes negative after treatment
    - examples include: rapid plasma reagin (RPR) and Venereal Disease Research Laboratory (VDRL)
- treponemal-specific tests
    - generally more complex and expensive but specific for syphilis
    - qualitative only and are reported as 'reactive' or 'non-reactive'
    - examples include: TP-EIA (*T. pallidum* enzyme immunoassay), TPHA (*T. pallidum* HaemAgglutination test)
    - the TP-EIA test has become increasingly popular in recent years

The testing algorithms for syphilis are complicated but typically involve a combination of a non-treponemal test with a treponemal-specific test.

Further points on non-treponemal tests

Causes of false positive non-treponemal (cardiolipin) tests:

- pregnancy
- SLE, anti-phospholipid syndrome
- tuberculosis
- leprosy
- malaria
- HIV

Example of test results

Positive non-treponemal test + positive treponemal test

- consistent with active syphilis infection

Positive non-treponemal test + negative treponemal test

- consistent with a false-positive syphilis result e.g. due to pregnancy or SLE (see list above)

Negative non-treponemal test + positive treponemal test :

- consistent with successfully treated syphilis



Next question

Textbooks

High-yield textbook

Extended textbook

Media



Syphilis

Osmosis - YouTube 3 0

Report broken media

Score: **16.7%**

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A 20-year-old woman who is 16 weeks pregnant presents with pain passing urine and an irritating rash. On examination, she has a tender, red, vesicular rash on her vulva. A urine dipstick shows both blood and white cells. What is the best treatment?

- Clotrimazole
- Cefalexin
- Oral aciclovir
- Topical aciclovir
- Fluconazole

Submit answer

Reference ranges

Score: **0%**

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Back to top

A 20-year-old woman who is 16 weeks pregnant presents with pain passing urine and an irritating rash. On examination, she has a tender, red, vesicular rash on her vulva. A urine dipstick shows both blood and white cells. What is the best treatment?

Clotrimazole	10%
Cefalexin	11%
Oral aciclovir	54%
Topical aciclovir	22%
Fluconazole	4%

This patient has genital herpes simplex virus (HSV). The guidelines recommend treatment with oral (or intravenous) aciclovir at any stage in pregnancy. Aciclovir is not licensed in pregnancy but is considered safe and not associated with birth defects. It is well tolerated in pregnancy. Paracetamol and topical lidocaine 2% gel can be used for symptomatic relief.

The primary purpose of treatment is to reduce the risk of transmission to the neonate at birth. The risk is much more considerable with primary genital herpes simplex within the final six weeks of pregnancy. Caesarian section should be the recommended mode of delivery for all women developing the first episode of genital HSV in the third trimester.

https:www.rcog.org.uk/globalassets/documents/guidelines/management-genital-herpes.pdf

Discuss (4) Improve

Next question

Herpes simplex virus

There are two strains of the herpes simplex virus (HSV) in humans: HSV-1 and HSV-2. Whilst it was previously thought HSV-1 accounted for oral lesions (cold sores) and HSV-2 for genital herpes it is now known there is considerable overlap

Features

- primary infection: may present with a severe gingivostomatitis

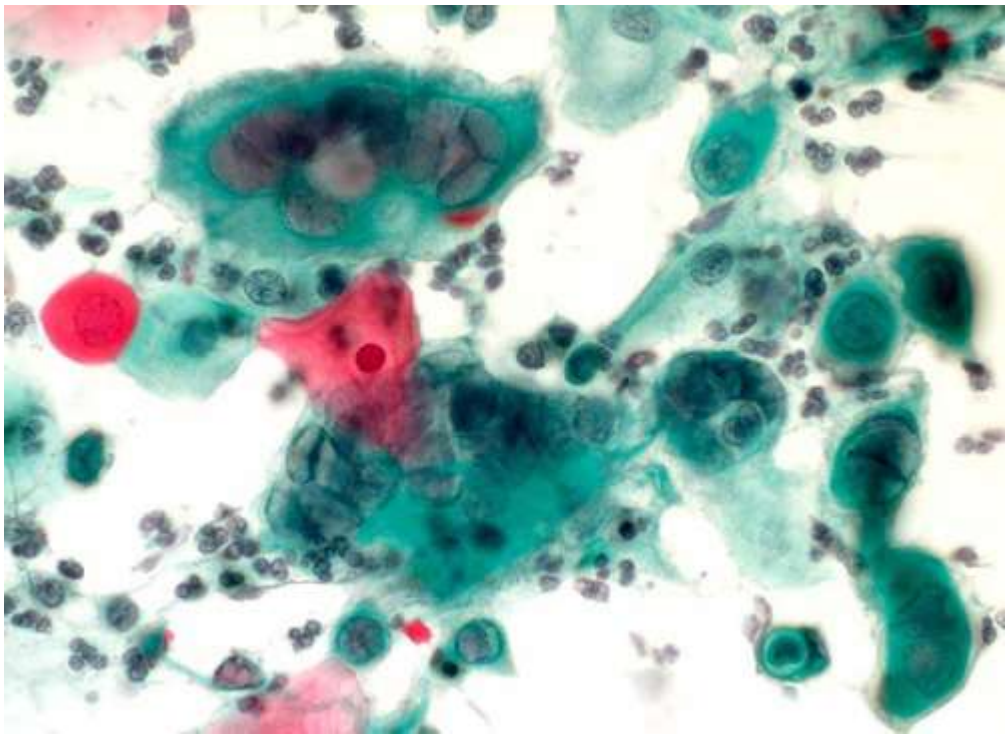
- cold sores
- painful genital ulceration

Management

- gingivostomatitis: oral aciclovir, chlorhexidine mouthwash
- cold sores: topical aciclovir although the evidence base for this is modest
- genital herpes: oral aciclovir. Some patients with frequent exacerbations may benefit from longer term aciclovir

Pregnancy

- elective caesarean section at term is advised if a primary attack of herpes occurs during pregnancy at greater than 28 weeks gestation
- women with recurrent herpes who are pregnant should be treated with suppressive therapy and be advised that the risk of transmission to their baby is low



© Image used on license from PathoPic

Pap smear. Multinucleated giant cells representing infection by the herpes simplex virus. Note the 3 M's; Multinucleation, Margination of the chromatin, Molding of the nuclei

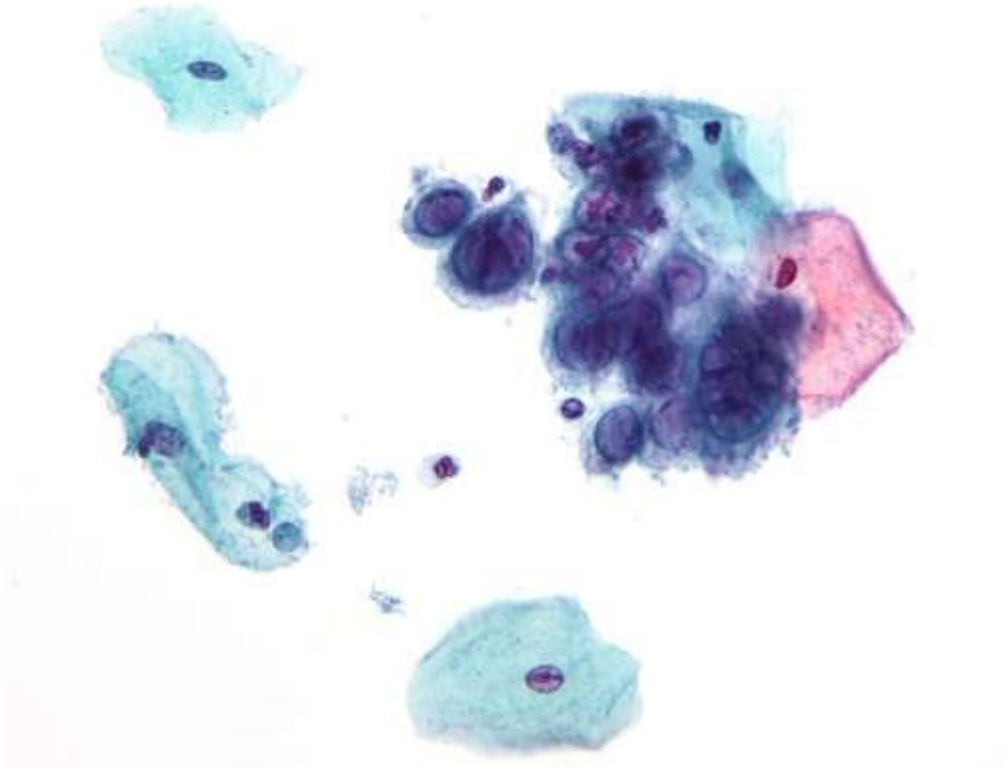


Image sourced from Wikipedia© Image used on license from PathoPic

Further Pap smear showing the cytopathic effect of HSV (multi-nucleation, ground glass & marginated chromatin)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

RCOG

6 3

2014 Management of Genital Herpes in Pregnancy

Suggest link

Report broken link

Media



Herpes (oral & genital)

Osmosis - YouTube 2 1

Report broken media

Score: **16.7%**

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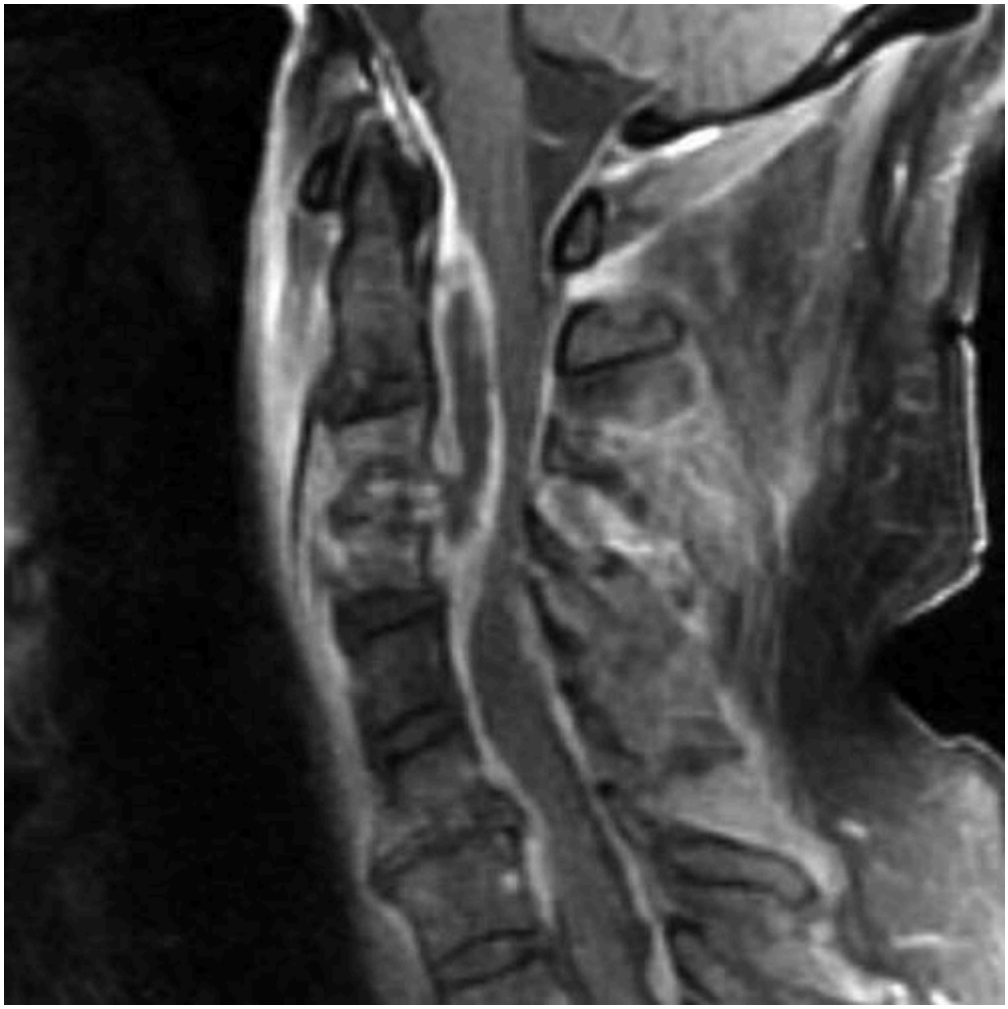
Back to top

A 54-year-old man presents with neck pain and feeling generally unwell. This has been getting progressively worse over the past two weeks and is now 'unbearable'. He feels hot and also complains of headaches.

He emigrated from Pakistan 30 years ago. He smokes 20 cigarattes/day and does not drink alcohol.

On examination pulse is 102/min, blood pressure 124/74 mmHg and temperature 37.9ºC. He has weakness in both arms

MRI of his cervical spine is shown below:



© Image used on license from Radiopaedia

What is the most likely diagnosis?

- Cervical disc prolapse
- Syringomyelia

- Cervical epidural abscess
- Meningitis
- Tuberculosis

Submit answer

Reference ranges

Score: **0%**

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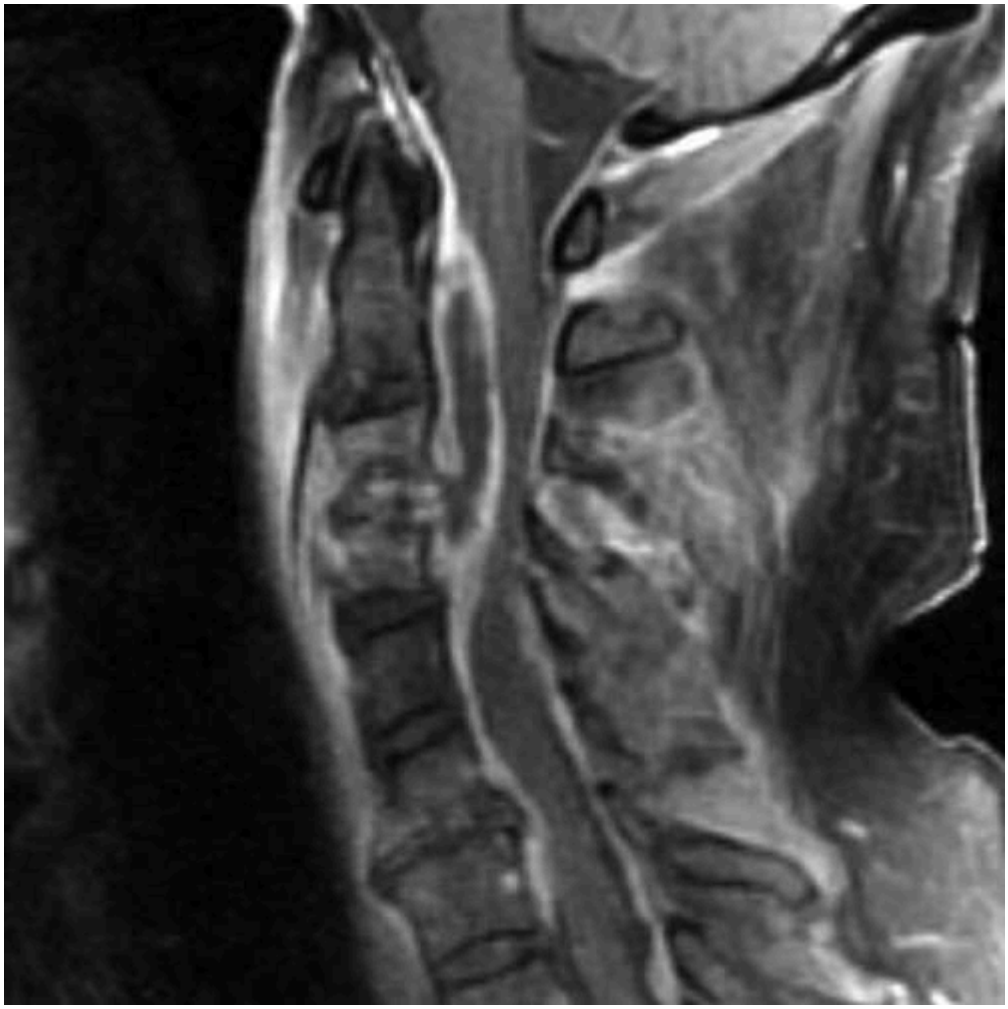
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He emigrated from Pakistan 30 years ago. He smokes 20 cigarattes/day and does not drink alcohol.

On examination pulse is 102/min, blood pressure 124/74 mmHg and temperature 37.9ºC. He has weakness in both arms

MRI of his cervical spine is shown below:



© Image used on license from Radiopaedia

What is the most likely diagnosis?

Cervical disc prolapse	3%
Syringomyelia	4%

Cervical epidural abscess	50%
Meningitis	0%
Tuberculosis	43%

MRI demonstrates an epidural collection with peripheral contrast enhancement. The cord is displaced posteriorly and to the right. Features are consistent with an epidural abscess. There are associated changes at the C3/4 level consistent with advanced discitis osteomyelitis.

Discuss (6) Improve

Next question >

Spinal epidural abscess ★

A spinal epidural abscess (SEA) is a collection of pus that is superficial to the dura mater (of the meninges) that cover the spinal cord. It is an emergency requiring urgent investigation and treatment to avoid progressive spinal cord damage.

> An abscess is a collection of pus encapsulated by a pyogenic membrane.

In SEA, bacteria enters the spinal epidural space by contiguous spread from adjacent structures (e.g. discitis), haematogenous spread from concomitant infection (e.g. bacteraemia from IVDU), or by direct infection (e.g. spinal surgery). Immunosuppression is another major risk factor, which may be caused by congenital immune disorders, acquired immune disorders (such as HIV, diabetes or alcoholism or by iatrogenic means (e.g. chemotherapy or steroids).

SEA is most typically bacterial and the most common causative micro-organism is *Staphylococcus aureus*.

Patients present with a combination:

- fever
- back pain
- focal neurological deficits according to the segment of the cord affected.

Investigations

- Bloods (including inflammatory markers, HIV, Hep B, Hep C, and preoperative blood tests (coagulation and group and screen))
- Blood cultures

- Infection screen (including chest x-ray and urine culture)*
- MRI whole spine (the entire spine is imaged since skip lesions may be present)

*If the primary source of infection is not clear, a wide search for sources requires investigations including echocardiography and dental x-rays.

All patients will require a long-term course of antibiotics which is at first broad spectrum but maybe later refined based on culture results. Patients with large or compressive abscesses, patients with significant or progressive neurological deficits or those who are not responding to antibiotics alone are considered for surgical evacuation of the abscess.



Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 29-year-old woman who is a former intravenous drug user is seen in the hepatology clinic. She is 12 weeks pregnant and has been referred as she is known to have chronic hepatitis C. Twelve months ago she was treated with pegylated interferon-alpha, ribavirin and a a protease inhibitor which failed to result in her clearing the hepatitis C virus.

What is the most appropriate way, if any, to reduce the risk of vertical transmission?

- Caesarean section
- Further course of antiviral therapy during pregnancy
- Advise against breastfeeding
- Antiviral therapy for the neonate for the first 4 weeks
- None of the above interventions is recommended

Submit answer

Reference ranges

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What is the most appropriate way, if any, to reduce the risk of vertical transmission?

Caesarean section	33%
Further course of antiviral therapy during pregnancy	9%
Advise against breastfeeding	11%
Antiviral therapy for the neonate for the first 4 weeks	17%
None of the above interventions is recommended	30%

Discuss (2) | Improve

Next question >

Hepatitis C and pregnancy

Hepatitis C is likely to become a significant public health problem in the UK in the next decade. It is thought around 200,000 people are chronically infected with the virus. At risk groups include intravenous drug users and patients who received a blood transfusion prior to 1991 (e.g. haemophiliacs).

Women in the UK are not currently screen for hepatitis C in the antenatal period.

Transmission risk

- the vertical transmission rate from mother to child is about 6%. The risk is higher if there is a high viral load or coexistent HIV

Management

- there are no definitive guidelines for the management of women with hepatitis C in pregnancy, the following is based on expert reviews
- standard drug therapy cannot be used in pregnancy due to concerns about teratogenicity
- HCV-positive pregnant women should be monitored throughout pregnancy
- HCV RNA and LFTs should be done as early as possible into prenatal care to assess the risk of transmission and degree of liver disease
- if pruritic or they develop jaundice, suspect intrahepatic cholestasis of pregnancy and perform LFTs.
- invasive procedures should be minimised in mother and fetus to prevent vertical transmission
- the evidence base surrounding the use of caesarean section vs vaginal delivery is inconclusive, but it is not currently routine practice to offer a caesarean section. Cochrane state: '*Currently there is no evidence from randomised controlled trials upon which to base any practice recommendations regarding planned caesarean section versus vaginal delivery for preventing mother to infant Hepatitis C Virus transmission*'
- exposure to maternal blood e.g. due to perineal tears significantly increases the risk of passing on the virus
- breastfeeding is not contraindicated in mothers with hepatitis C



Next question

Textbooks

High-yield textbook

Extended textbook

Links

American Journal of Perinatology

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2013 Hepatitis C Virus in Pregnancy

Suggest link Report broken link

Score: **16.7%**

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A 45-year-old female presents with a history of headache and generalised malaise for the last two months. She describes her headaches as being a tight band like sensation which is present almost throughout the day and causes significant difficulty in sleeping at night. She also mentions occasional episodes of vomiting along with low-grade fever and weight loss of about 7 kg over the same duration of time. She suffers from generalised anxiety disorder and takes 0.5mg alprazolam TDS. She returned from Dubai 9 months ago where she had been spending her holidays with her family.

On examination, she has a fever of 37.5°C and a pulse of 105bpm. She appears slightly disoriented with a tendency to speak out of context but is otherwise cooperative.

There is diplopia on right sided gaze and mild neck stiffness, but the remaining clinical examination is essentially unremarkable.

Lab reports reveal:

Hb	115 g/l
Platelets	340 * 10^9/l
WBC	9.0 * 10^9/l

Na^+	137 mmol/l
K^+	4.2 mmol/l
Urea	5.9 mmol/l
Creatinine	102 µmol/l
Glucose	7.0 mmol/l
ESR	87 mm/hr

MRI shows meningeal enhancement but no evidence of any parenchymal lesions.

CSF examination reveals:

Opening Pressure	Normal
Appearance	Turbid
Protein	3.2g/L (0.2 0.4 g/L)
Glucose	2.7 mmol/l
Lymphocytes	371/mm³

Neutrophils	42/mm³
ZN staining	No acid fast bacilli detected

Which of the following is the most appropriate treatment option?

- IV acyclovir
- IV ceftriaxone
- Isoniazid, rifampicin, pyrazinamide and dexamethasone
- IV benzylpenicillin
- Artemether/lumefantrine

Submit answer

Reference ranges

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Glucose	2.7 mmol/l
Lymphocytes	371/mm³

Neutrophils	42/mm³
ZN staining	No acid fast bacilli detected

Which of the following is the most appropriate treatment option?

IV acyclovir	23%
IV ceftriaxone	12%
Isoniazid, rifampicin, pyrazinamide and dexamethasone	54%
IV benzylpenicillin	5%
Artemether/lumefantrine	5%

The 2-month history of headache, generalised malaise, low-grade fever and weight loss all go in favour of a diagnosis of tuberculous meningitis. This diagnosis is also supported by the examination findings of diplopia neck stiffness.

The complete blood count does not favour a bacterial infection as one would expect a leucocytosis. The raised ESR is also supportive of TBM.

The CSF shows a predominant lymphocytosis with greatly raised protein and significantly low glucose which once again goes with tuberculosis.

TBM typically commences with symptoms of a vague headache, lassitude, anorexia and vomiting. Acute meningitis may occur but it is uncommon. The development of meningitic signs often takes weeks. Common signs include diplopia, papilloedema and hemiparesis and seizures are also often commonly seen. Treatment is generally commenced on a presumptive basis since multiple CSF cultures are often required and it may take weeks before culture reports are available. Staining with ZN staining is often negative and PCR for mycobacterium tuberculosis is helpful but is often negative too and repeat PCR is often needed.

Treatment is with isoniazid, rifampicin, pyrazinamide and steroids. Ethambutol should be avoided due to eye complications. Details regarding antimicrobial therapy and duration of treatment are available at http://emedicine.medscape.com/article/1166190-treatment#aw2aab6b6b2

Discuss (12) Improve

Next question ›

Meningitis: CSF analysis

The table below summarises the characteristic cerebrospinal fluid (CSF) findings in meningitis:

	Bacterial	Viral	Tuberculous	Fungal
Appearance	Cloudy	Clear/cloudy	Slight cloudy, fibrin web	Cloudy
Glucose	Low (< 1/2 plasma)	60-80% of plasma glucose*	Low (< 1/2 plasma)	Low
Protein	High (> 1 g/l)	Normal/raised	High (> 1 g/l)	High
White cells	10 - 5,000 polymorphs/mm^3	15 - 1,000 lymphocytes/mm^3	30 - 300 lymphocytes/mm^3	20 - 200 lymphocytes/mm^3

The Ziehl-Neelsen stain is only 20% sensitive in the detection of tuberculous meningitis and therefore PCR is sometimes used (sensitivity = 75%)

*mumps is unusual in being associated with a low glucose level in a proportion of cases. A low glucose may also be seen in herpes encephalitis

AI + Q 123

Next question >

Textbooks

High-yield textbook

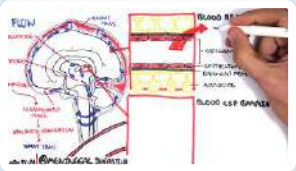
Extended textbook

Media



Meningitis

Osmosis - YouTube 3 0



(Bacterial) Meningitis Pathophysiology

Armando Hasudungan - YouTube 2 3

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Score: **16.7%**

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Back to top

A 44-year-old woman with a background of asthma who has required frequent use of oral corticosteroids is inquiring about vaccinations available to her. Which one of the following should be recommended?

- 23-valent unconjugated pneumococcal polysaccharide vaccine and influenza vaccine
- 13-valent conjugated pneumococcal polysaccharide vaccine only
- 23-valent unconjugated pneumococcal polysaccharide vaccine only
- 13-valent conjugated pneumococcal polysaccharide vaccine and influenza vaccine
- Influenza vaccine only

Submit answer

Reference ranges

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Back to top

A 44-year-old woman with a background of asthma who has required frequent use of oral corticosteroids is inquiring about vaccinations available to her. Which one of the following should be recommended?

23-valent unconjugated pneumococcal polysaccharide vaccine and influenza vaccine	47%
13-valent conjugated pneumococcal polysaccharide vaccine only	3%
23-valent unconjugated pneumococcal polysaccharide vaccine only	5%
13-valent conjugated pneumococcal polysaccharide vaccine and influenza vaccine	31%
Influenza vaccine only	15%

Patients with chronic respiratory conditions including asthma requiring frequent use of oral steroids should be offered both the 23-valent unconjugated pneumococcal polysaccharide vaccine and the influenza vaccine. In this population, the pneumococcal vaccine is usually given as a once off but may be required 5 yearly depending on the underlying condition and the age of the patient. The influenza vaccine, however, is given annually.

The 13-valent conjugated pneumococcal polysaccharide vaccine is routinely given to infants as it has been found to be more effective given their less mature immune systems.

Discuss (6) | Improve

Next question >

Pneumococcal vaccine

There are two type of pneumococcal vaccine currently in use:

- pneumococcal conjugate vaccine (PCV)
- pneumococcal polysaccharide vaccine (PPV)

The PCV is given to children as part of their routine immunisations (at 3 and 12-13 months).

The PPV is offered to all adults over the age of 65 years, to patients with chronic conditions such as COPD and to those who have had a splenectomy (see below).

Groups who should be vaccinated:

- asplenia or splenic dysfunction
- chronic respiratory disease: COPD, bronchiectasis, cystic fibrosis, interstitial lung disease. Asthma is only included if 'it requires the use of oral steroids at a dose sufficient to act as a significant immunosuppressant'
- chronic heart disease: ischaemic heart disease if requiring medication or follow-up, heart failure, congenital heart disease. Controlled hypertension is not an indication for vaccination
- chronic kidney disease
- chronic liver disease: including cirrhosis and chronic hepatitis
- diabetes mellitus if requiring medication
- immunosuppression (either due to disease or treatment). This includes patients with any stage of HIV infection
- cochlear implants
- patients with cerebrospinal fluid leaks

Adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years.



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Greenbook



Pneumococcal

Clinical Knowledge Summaries

4 0

Pneumococcal vaccine

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Score: **16.7%**

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Back to top

A 39-year-old female with end-stage renal failure secondary to IgA nephropathy is admitted to hospital. She was discharged 3 days earlier following an admission due to collapse in the context of hyperkalaemia (Potassium 7.8 mmol/l). She has been transferred to the medical assessment unit having developed rigors on dialysis earlier today.

Bloods taken at the end of dialysis reveal:

White cell count	15.2 *109/l
Sodium	134 mmol/l
Potassium	2.1 mmol/l
Urea	10.6 mmol/l
Creatinine	400 µmol/l
C-reactive protein (CRP)	119 mg/dL

What is the immediate priority in her management?

- Replace potassium (K+)
- Replace magnesium (Mg2+)
- Intravenous antibiotics
- Intravenous calcium gluconate
- Insulin/dextrose infusion

Submit answer

Reference ranges

Score: **0%**

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What is the immediate priority in her management?

Replace potassium (K+)	34%
Replace magnesium (Mg2+)	19%
Intravenous antibiotics	36%
Intravenous calcium gluconate	10%
Insulin/dextrose infusion	1%

The patient is septic and infection is her most imminent threat. Post-dialysis bloods are often misleading in terms of electrolytes as it takes 30 minutes for intracellular and extracellular compartments to equilibrate. Assuming that the patients pre-dialysis bloods showed a high-normal K+ (which would fit with her recent hyperkalaemia presentation) then her total body K+ will be much higher than the post-dialysis sample suggests. The bloods should be repeated to confirm this but over-zealous correction of K+ in this situation can be dangerous.

Magnesium is important to correct in the presence of hypokalaemia, however, we do not think this patient genuinely has low potassium. Intravenous calcium gluconate and insulin/dextrose infusion are both treatments for hyperkalaemia and therefore are not appropriate.

Sepsis

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to an infection. Sepsis is increasingly recognised as an important cause of mortality in the UK and there has been increasing efforts recently to improve the care of patients who present with sepsis.

How sepsis is classified has changed in recent years - the Surviving Sepsis Guidelines were updated in 2017.

The new guidelines recognise the following terms:

- **sepsis**: life-threatening organ dysfunction caused by a dysregulated host response to infection
- **septic shock**: a more severe form sepsis, technically defined as *'in which circulatory, cellular, and metabolic abnormalities are associated with a greater risk of mortality than with sepsis alone'**

The old category of severe sepsis is no longer used.

The term 'systemic inflammatory response syndrome (SIRS)' has also fallen out of favour. Adult patients outside of ICU with suspected infection are identified as being at heightened risk of mortality if they have quickSOFA (qSOFA) score meeting >= 2 of the following criteria: respiratory rate of 22/min or greater, altered mentation, or systolic blood pressure of 100mmHg or less

qSOFA score
Respiratory rate > 22/min
Altered mentation
Systolic blood pressure < 100 mm Hg

Within an ICU setting a full SOFA** score is often used. Details can be found at the bottom of the notes.

Management

NICE released its own guidelines in 2016. These focussed on the risk stratification and management of patients with suspected.

For risk stratification NICE recommend using the following criteria:

Red flag criteria	Amber flag criteria
• Responds only to voice or pain/ unresponsive • Acute confusional state • Systolic B.P <= 90 mmHg (or drop >40 from normal) • Heart rate > 130 per minute • Respiratory rate >= 25 per minute • Needs oxygen to keep SpO2 >=92% • Non-blanching rash, mottled/ ashen/ cyanotic • Not passed urine in last 18 h/ UO < 0.5 ml/kg/hr • Lactate >=2 mmol/l • Recent chemotherapy	• Relatives concerned about mental status • Acute deterioration in functional ability • Immunosuppressed • Trauma/ surgery/ procedure in last 6 weeks • Respiratory rate 21-24 • Systolic B.P 91-100 mmHg • Heart rate 91-130 OR new dysrhythmia • Not passed urine in last 12-18 hours • Temperature < 36ºC • Clinical signs of wound, device or skin infection

Clearly, the underlying cause of the patient's sepsis needs to be identified and treated and the patient supported regardless of the cause or severity. If however any of the red flags are present the 'sepsis six' should be started straight away:

- 1. Administer oxygen: Aim to keep saturations > 94% (88-92% if at risk of CO2 retention e.g. COPD)
- 2. Take blood cultures
- 3. Give broad-spectrum antibiotics
- 4. Give intravenous fluid challenges
    - NICE recommend a bolus of 500ml crystalloid over less than 15 minutes
- 5. Measure serum lactate
- 6. Measure accurate hourly urine output

*these patients can be clinically identified by a vasopressor requirement to maintain a MAP ≥ 65mmHg and serum lactate >2mmol/L in the absence of hypovolemia

**To help identify and categorise patients the Sequential (Sepsis-Related) Organ Failure Assessment Score (SOFA) is increasingly used. The score grades abnormality by organ system and accounts for clinical interventions. However, laboratory variables, namely, PaO_2, platelet count, creatinine level, and bilirubin level, are needed for full computation.

SOFA Score

System	Score 0	Score 1	Score 2	Score 3	Score 4
PaO_2 /Fl O_2	>400	<400	<300	<200	<100
Platelets x10^3 microlitres	>150	<150	<100	<50	<20
Bilirubin µmol/L	20	20-32	33-101	102-204	>204
Cardiovascular	MAP >70mmHg	MAP 70mmHg	Dopamine <5 or dobutamine (any dose)	Dopamine 5.1-15 or epinephrine 0.1 or norepinephrine 0.1	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1
GCS	15	13-14	10-12	6-9	<6
Creatinine µmol/L	<110	110-170	171-299	300-440	>440
Urine output ml/day	>500	>500	>500	<500	<200

A SOFA score of 2 or more reflects an overall mortality risk of approximately 10% in a general hospital population with suspected infection. Even patients presenting with modest dysfunction can deteriorate further, emphasizing the seriousness of this condition and the need for prompt and appropriate intervention, if not already being instituted.



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane — 4 5

Sepsis Definitions and Diagnosis

European Society of Intensive Care Medicine — 2 3

Sepsis-3

Sepsis Trust — 0 1

Sepsis clinical toolkit

Journal of American Medical Association — 4 4

The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)

NICE — 8 7

2016 Sepsis: recognition, diagnosis and early management

Royal College of Physicians — 3 3

Sepsis: recognition and treatment

Royal College of Physicians — 4 3

2011 Sepsis: an update for physicians

Surviving Sepsis Campaign — 6 3

Guidelines

Core EM — 1 0

Surviving Sepsis 2017 Guidelines Overview

Suggest link Report broken link

Media



An introduction to the SEPSIS pathway (detect it early and treat it early)

Armando Hasudungan - YouTube

2 0



Understanding Sepsis (Sepsis Explained Clearly)

Rhesus Medicine - YouTube

0 0

Report broken media

Score: **16.7%**

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Back to top

A 23-year-old student presented to his GP with fever and epistaxis. The fever had started 6 days earlier. It had been associated with a headache and malaise. Following clinical examination his doctor prescribed him amoxicillin which he took for 3 days but without any improvement. He later became so ill that he refused to eat and started to vomit.

His history included mumps at the age of nine. His father had been diagnosed with liver cirrhosis 2 years ago due to excessive alcohol consumption. He is a regular smoker for the last 10 years and lives in the dorm. Two weeks earlier he had been in rural Central Africa as a reporter covering the conflict there. He denied being vaccinated for any disease before going there but did take anti-malarial tablets.

On examination he appeared ill. His temperature was 38ºC. He was jaundiced. Eye examination revealed conjunctival hemorrhages on both eyes. All other systems were normal.

The following investigations had been requested:

Hb	13 g/dl
platelets	170 * 10^9/l
WBC	4 * 10^9/l
MCV	85 fl
MCH	0.4 fmol/cell
MCHC	20 mmol/l
Na+	135 mmol/l
K+	4 mmol/l
Creatinine	80 µmol/l
Urea	3 mmol/l
ESR	60 mm/hr
Alkaline phosphatase	100 IU/l
Alanine transaminase	400 IU/l
Aspartate transaminase	200 IU/l
Bilirubin	25 µmol/l (direct 18 umol/l)
Serum albumin	40 g/l
Prothrombin time	prolonged
Partial thrompoblastin time	prolonged
Urine analysis	Albumin + ,acetone ++ ,bile pigment ++,urinary urobilinogen +

Thick Blood film for malaria	negative

What is the most likely diagnosis?

- Yellow fever
- Primary sclerosing cholangitis
- Leptospirosis
- Falciparum malaria
- Dengue fever

Submit answer

Reference ranges

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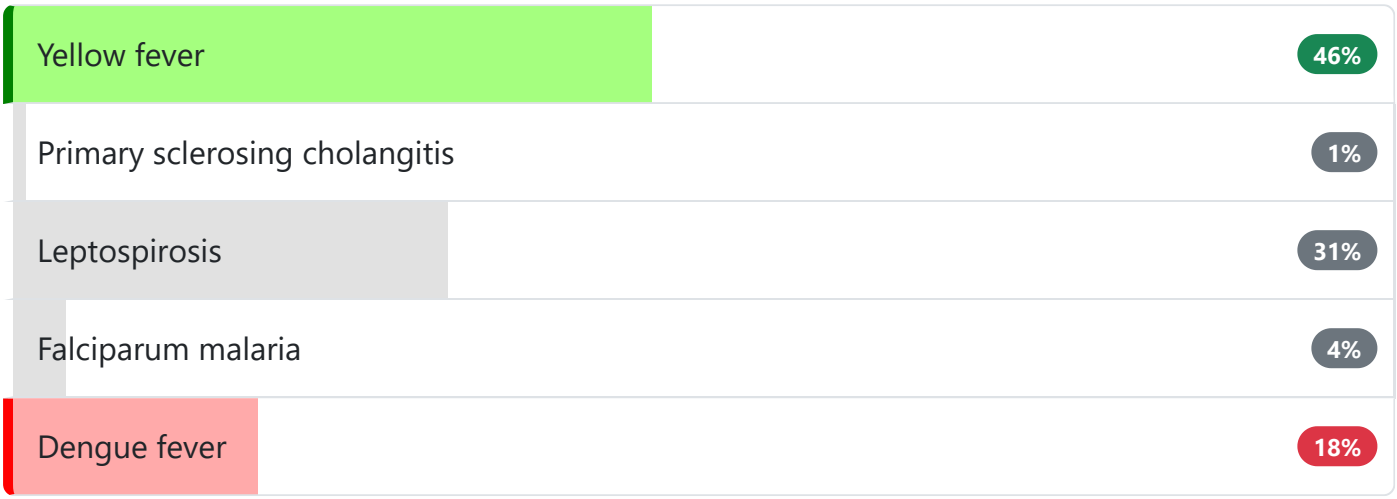
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Alanine transaminase	400 IU/l
Aspartate transaminase	200 IU/l
Bilirubin	25 µmol/l (direct 18 umol/l)
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Partial thrompoblastin time	prolonged
Urine analysis	Albumin + ,acetone ++ ,bile pigment ++,urinary urobilinogen +

Thick Blood film for malaria	negative

What is the most likely diagnosis?



Yellow fever is endemic around the equator mainly in Africa. It is transmitted by the Aedes mosquitoes there. Characteristically the patient presents with fever and constitutional symptoms. Jaundice is a main clinical sign associated most of the times with conjunctival injection. Other important clinical signs are relative bradycardia, and hemorrhagic diathesis. The cause of death is due to multi-organ failure.

Blood tests generally show leucopenia with predominant neutropenia but this is not specific. Liver enzymes are elevated most of the time. Diagnosis depends on a high degree of suspicion for the disease and but also more important on serology which is highly specific for this lethal condition.

Treatment is mainly supportive.

Primary sclerosing cholangitis could present with fever and jaundice. This would happen in the context of ascending cholangitis (fever + pain + jaundice) with a long history of abdominal discomfort. Liver profile would reflect obstructive changes.

Malaria should be excluded in any patient who had traveled to an endemic area and developed fever. The picture in our patient was not typical and the blood film had been negative for malaria parasites. However one has to keep it in mind if the clinical picture fits. In that case one needs to repeat the blood film and to order serology.

Leptospirosis is a consideration here. But it usually responds to antibiotics. Liver enzymes would generally be more mildly elevated.

Dengue fever mainly presents with headache, fever and rash. It is mainly endemic in South East Asia.

Discuss (10) Improve

Next question

Yellow fever

Type of viral haemorrhagic fever (also dengue fever, Lassa fever, Ebola).

Basics

- zoonotic infection: spread by *Aedes* mosquitos
- incubation period = 2 - 14 days

Features

- may cause mild flu-like illness lasting less than one week
- classic description involves sudden onset of high fever, rigors, nausea & vomiting. Bradycardia may develop. A brief remission is followed by jaundice, haematemesis, oliguria
- if severe jaundice, haematemesis may occur
- Councilman bodies (inclusion bodies) may be seen in the hepatocytes

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 60 year old male who is a malnourished alcoholic presents with a chronic cough for the past 6 weeks associated with a low grade fever. The cough is productive of purulent sputum.

Six months previously he had been diagnosed with early stage non-Hodgkin's lymphoma, which had responded well to chemotherapy (doxorubicin, bleomycin, vinblastine, and prednisolone).

On examination his temperature is 37.8ºC, blood pressure 140/80 mmHg, and his pulse is 96/minute and regular. Auscultation of the chest reveals absence of breath sounds over the left middle lung field. Chest x-ray confirms left upper lobar consolidation.

The following investigations were ordered:

Hb	12 g/dl
Platelets	180 * 10^9/l
WBC	7 * 10^9/l
MCV	85 fl
Na+	140 mmol/l
K+	5 mmol/l
Creatinine	90 µmol/l
Urea	5 mmol/l
CRP	50 mg/l

Sputum stains partially acid fast bacilli with branching rods

What is the most suitable initial management of this patient?

- Metronidazole + ampicillin
- Clarithromycin
- Ceftriaxone + clarithromycin
- Trimethoprim/sulfamethoxazole + amikacin + ceftriaxone
- Isoniazid + rifampin + pyrazinamide + ethambutol

Submit answer

Reference ranges

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Sputum stains partially acid fast bacilli with branching rods

What is the most suitable initial management of this patient?

Metronidazole + ampicillin	6%
Clarithromycin	7%
Ceftriaxone + clarithromycin	10%
Trimethoprim/sulfamethoxazole + amikacin + ceftriaxone	27%
Isoniazid + rifampin + pyrazinamide + ethambutol	50%

Nocardiosis typically occurs in immunocompromised patients with pulmonary involvement and in

most cases with CNS or skin dissemination.

The clue here is the acid-fast stain which shows partially acid fast branching rods organism in contrast to mycobacterium which does fully stain.

Treatment of immunocompromised patients consists of multiple regimens. But mainly all of them involve Trimethoprim/sulfamethoxazole.

Nocardia species are ubiquitous soil organisms that often infect patients who are immunosuppressed, have pulmonary disease, or have a history of surgery or trauma. It is a Gram positive, acid fast bacilli.

Discuss (8) | Improve

Next question >

Actinomyces and *Nocardia*

Both *Actinomyces* and *Nocardia* are Gram-positive rods that form fungus-like branched networks of hyphae-like filaments.

Actinomyces israelii

Basics

- chronic, progressive granulomatous disease caused by filamentous Gram-positive anaerobic bacteria from the *Actinomycetaceae* family.
- typically causes oral/facial abscesses with sulphur granules in sinus tracts
- may also cause an abdominal mass e.g. in the right iliac fossa

Actinomyces are commensal bacteria that become pathogenic when a mucosal barrier is breached.

The disease most commonly occurs in the head and neck, although it may also occur in the abdominal cavity and in the thorax.

The mass will often enlarge across tissue planes with the formation of multiple sinus tracts.

Abdominopelvic actinomycosis occurs most frequently in individuals that have had appendicitis (65%).

Pathology

- On histological examination Gram-positive organisms and evidence of sulphur granules.
- Sulphur granules are colonies of organisms that appear as round or oval basophilic masses.
- They are also seen in other conditions such as nocardiosis

Treatment

- Long-term antibiotic therapy usually with penicillin
- Surgical resection is indicated for extensive necrotic tissue, non-healing sinus tracts, abscesses or where biopsy is needed to exclude malignancy.

References

Wong V, Turmezei T and Weston V. Actinomycosis. *BMJ* 2011;343d6099.

Nocardia

Basics

- typically causes pneumonia in immunocompromised patients
- may also cause brain abscesses



Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 27 year old Caucasian man has just returned from a 12 week trip to Ivory Coast where he was working as a missionary. He had not consulted a travel clinic for immunisations and had taken no malaria prophylaxis. During his trip he slept in a hammock outside and he took a daily wash in the river near the church where he was working. For the last few days of his trip he felt very unwell. He developed fevers, muscle aches and a headache, however this was improving and he had been feeling much better when he arrived home in the UK.

He has been home for 24 hours. Today he felt very much worse and presented to A and E.

On admission:

- Temperature: 39.1C
- Blood pressure: 89/61mmHg
- Pulse: 68 / min
- Respiratory rate: 26 / min
- Oxygen saturations: 97% on room air.

His initial investigations show:

Hb	11.1 g/dl
Platelets	61 * 10^9/l
WBC	3.3 * 10^9/l
Neutrophils	2.6 * 10^9/l
Lymphocytes	0.6 * 10^9/l

Na^+	146 mmol/l
K^+	4.1 mmol/l
Bicarbonate	16 mmol/l
Urea	17.4 mmol/l
Creatinine	321 mol/l

Bilirubin	88 mol/l
ALP	322 u/l
ALT	821 u/l
γGT	421 u/l
Albumin	29 g/l

Following discussion with the ID consultant on call, he is isolated while a blood sample is sent to Public Health Englands imported fever service. He received empirical therapy for Malaria with Artesunate and for sepsis with Piperacillin/Tazobactam and Gentamicin, however he rapidly deteriorated and died within 24 hours of admission. Following discussion with Public Health England, who confirm that there was no evidence of a nosocomially transmitted Viral Haemorrhagic Fever, he has a post mortem examination which reveals Councilman bodies in his liver.

What would likely have prevented his death?

- ○ Sleeping under an insecticide treated bed net ×
- ○ Avoiding bathing in the river ×
- ○ Live attenuated Yellow Fever vaccine ×
- ○ Prophylaxis with doxycycline ×
- ○ Avoiding unprotected sex ×

Submit answer

Reference ranges

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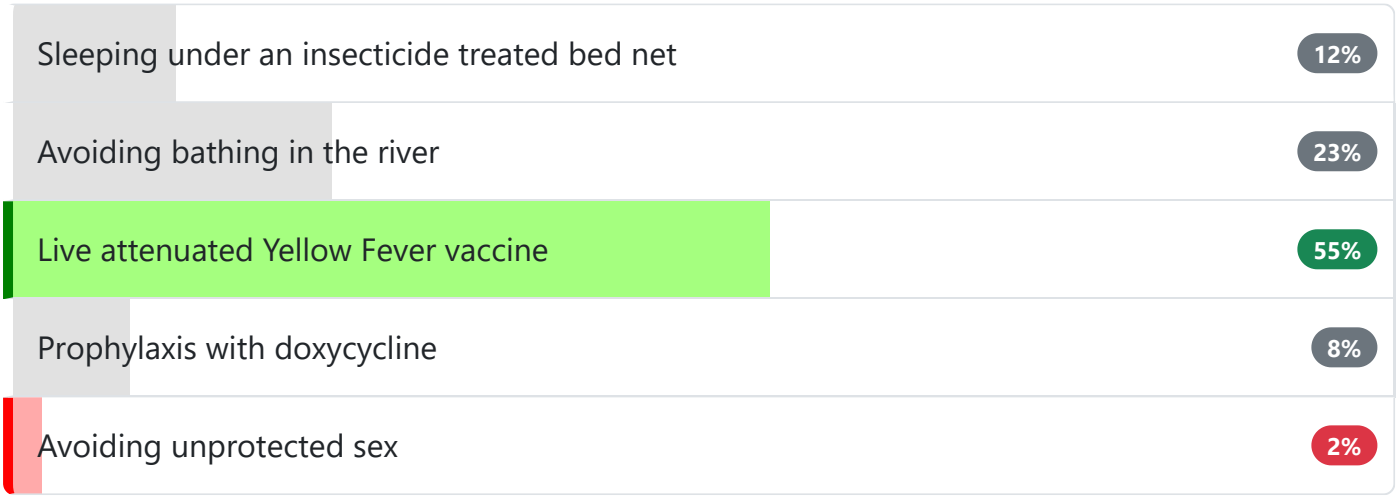
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What would likely have prevented his death?



This is yellow fever as suggested by:

- Travel to endemic area (West Africa and Central America)
- Fever, with initial resolution
- Progression to jaundice and renal failure

For exam purposes Councilman bodies (eosinophilic inclusion in the liver on post mortem) are diagnostic of yellow fever, although they can occasionally be seen in other Viral Haemorrhagic Fevers such as Crimean Congo Haemorrhagic Fever, (but this is nosocomially spread and therefore not the case in this example.

As such the vaccination is the only intervention which could have prevented his death.

Discuss (11) Improve

Next question >

Yellow fever ★

Type of viral haemorrhagic fever (also dengue fever, Lassa fever, Ebola).

Basics

- zoonotic infection: spread by *Aedes* mosquitos
- incubation period = 2 - 14 days

Features

- may cause mild flu-like illness lasting less than one week
- classic description involves sudden onset of high fever, rigors, nausea & vomiting. Bradycardia may develop. A brief remission is followed by jaundice, haematemesis, oliguria
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Next question

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High-yield textbook

Extended textbook

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Back to top

A 37 year old Caucasian male attends an outpatient clinic. He had recently spent three months in Jamaica on a beach-side yoga retreat. He only drank bottled water and ate well cooked vegetarian food. He ensured that any raw vegetables were peeled. He returned home three weeks ago.

During his final week in Jamaica he noticed an itchy spot develop on the side of his ankle. The rash has gradually spread from the original spot slowly 'like a snake up the side of his foot'. The lesion is currently 11cm long and is a raised itchy serpiginous linear lesion.

He is otherwise well.

What is the causative organism?

- *Strongyloides stercoralis* ✗
- *Ancylostoma braziliense* ✗
- *Necator americanus* ✗
- *Ascaris lumbricoides* ✗
- *Schistosomiasis mansoni* ✗

Submit answer

Reference ranges

Score: **16.7%**

1 ✗
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A 37 year old Caucasian male attends an outpatient clinic. He had recently spent three months in Jamaica on a beach-side yoga retreat. He only drank bottled water and ate well cooked vegetarian food. He ensured that any raw vegetables were peeled. He returned home three weeks ago.

During his final week in Jamaica he noticed an itchy spot develop on the side of his ankle. The rash has gradually spread from the original spot slowly 'like a snake up the side of his foot'. The lesion is currently 11cm long and is a raised itchy serpiginous linear lesion.

He is otherwise well.

What is the causative organism?

Strongyloides stercoralis	49%
Ancylostoma braziliense	30%
Necator americanus	6%
Ascaris lumbricoides	10%
Schistosomiasis mansoni	4%

The correct answer in this case is ***Ancylostoma braziliense***. The patient's presentation of a raised, itchy serpiginous linear lesion that has gradually spread 'like a snake' up the side of his foot is characteristic of cutaneous larva migrans (CLM), also known as creeping eruption. CLM is caused by the larval form of hookworms, most commonly *Ancylostoma braziliense*, which are found in tropical and subtropical regions such as Jamaica. The larvae penetrate the skin, usually through contact with contaminated soil or sand, and migrate within the epidermis, causing an intensely pruritic inflammatory reaction.

Strongyloides stercoralis is an incorrect option because this nematode causes strongyloidiasis, which typically presents with gastrointestinal symptoms such as abdominal pain and diarrhea. Skin involvement in strongyloidiasis can occur but usually manifests as pruritic papules or urticaria rather than the serpiginous linear lesion seen in this case.

Necator americanus is another species of hookworm, but it primarily causes human infection through intestinal penetration rather than cutaneous manifestations like CLM. Infection with *Necator americanus* can lead to iron-deficiency anemia due to blood loss from attachment sites in the small intestine.

Ascaris lumbricoides is a roundworm that causes ascariasis, one of the most common helminthic infections worldwide. Ascariasis typically presents with gastrointestinal symptoms such as abdominal pain and bloating; however, during the larval migration phase through the lungs, patients may experience coughing and wheezing. Cutaneous manifestations are not typical in ascariasis.

Schistosomiasis mansoni is a blood fluke that causes schistosomiasis, which is characterized by granulomatous inflammation in response to the eggs of the parasite. The primary manifestation of schistosomiasis is hepatosplenic involvement, with symptoms such as abdominal pain and hepatomegaly. Cutaneous manifestations can occur during the cercarial penetration phase (swimmer's itch), but this is typically a localized pruritic papular rash rather than the serpiginous linear lesion seen in this case.

Discuss (5) | Improve

Next question ›

Cutaneous larva migrans ★

Cutaneous larva migrans is a dermatological condition prevalent in tropical and subtropical regions, largely attributable to cutaneous penetration and subsequent migration of nematode larvae, primarily from the *Ancylostoma* genus (e.g. *Ancyclostoma braziliense*). Typically, the transmission vectors are faecal-contaminated soil or sand, posing significant risks to individuals with a history of barefoot beach visits or direct soil contact.

The clinical presentation typically involves an intensely pruritic, 'creeping', serpiginous, erythematous cutaneous eruption that advances over time. Symptoms can last for weeks to months, potentially leading to secondary bacterial infection due to excessive scratching. Diagnosis is typically clinical, based on exposure history and characteristic skin manifestations.



Treatment options revolve around anthelmintic agents, such as ivermectin or albendazole. Topical therapy with thiabendazole can also be effective, although it's generally less preferred due to a lower efficacy rate and higher side effect profile.

Prevention strategies largely involve patient education about appropriate protective measures, particularly avoiding direct skin contact with potentially contaminated soil. Given the zoonotic

nature of cutaneous larva migrans, public health measures for animal defecation control can also contribute to reducing the prevalence of this condition.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Center for Disease Control

3 2

Cutaneous Larva Migrans

Suggest link

Report broken link

Score: **16.7%**

1 ✗
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A 24-year-old Somali woman attends her booking appointment in the UK for her first pregnancy. Screening tests reveal that she is HIV positive. She is asymptomatic.

Her viral load is 150 000 copies/ml and her CD4 count is 523 cells/mm^3. No viral resistance is detected. Her hepatitis serology is negative. Her husband consequently tests negative for HIV.

She is commenced on triple antiretroviral therapy (ART) with zidovudine, lamivudine and lopinavir/ritonavir. By 36 weeks her viral load is undetectable at <20 copies/ml.

Which of the following statements is true regarding her ongoing management?

- She should have an elective caesarean section and continue to take her ART whilst breast-feeding. ✗
- She should have a vaginal delivery and continue to take ART whilst breast feeding. ✗
- She should have an elective caesarean section. ART should be continued. ✗
- She should have a vaginal delivery and formula feed. ART can be discontinued. ✗
- She should have a vaginal delivery and formula feed. ART should be continued. ✗

Submit answer

Reference ranges

Score: **16.7%**

1 ✗
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She is commenced on triple antiretroviral therapy (ART) with zidovudine, lamivudine and lopinavir/ritonavir. By 36 weeks her viral load is undetectable at <20 copies/ml.

Which of the following statements is true regarding her ongoing management?

She should have an elective caesarean section and continue to take her ART whilst breast-feeding.	11%
She should have a vaginal delivery and continue to take ART whilst breast feeding.	16%
She should have an elective caesarean section. ART should be continued.	17%
She should have a vaginal delivery and formula feed. ART can be discontinued.	3%
She should have a vaginal delivery and formula feed. ART should be continued.	52%

In this case, as her viral load is less than 50 copies/ml she may have a vaginal delivery. Formula feeding is recommended in the UK to all babies with HIV +ve mothers. In this case although her CD4 count is >350 cells/mm^3 her ART should be continued to reduce the risk of onward transmission to her husband.

Discuss (10) | Improve

Next question >

HIV and pregnancy

With the increased incidence of HIV infection amongst the heterosexual population there are an increasing number of HIV positive women giving birth in the UK. In London the incidence may be as high as 0.4% of pregnant women. The aim of treating HIV positive women during pregnancy is to minimise harm to both the mother and fetus, and to reduce the chance of vertical transmission.

Guidelines regularly change on this subject and most recent guidelines can be found using the links provided.

Factors which reduce vertical transmission (from 25-30% to 2%)

- maternal antiretroviral therapy
- mode of delivery (caesarean section)
- neonatal antiretroviral therapy
- infant feeding (bottle feeding)

Screening

- NICE guidelines recommend offering HIV screening to all pregnant women

Antiretroviral therapy

- all pregnant women should be offered antiretroviral therapy regardless of whether they were taking it previously

Mode of delivery

- vaginal delivery is recommended if viral load is less than 50 copies/ml at 36 weeks, otherwise caesarian section is recommended
- a zidovudine infusion should be started four hours before beginning the caesarean section

Neonatal antiretroviral therapy

- zidovudine is usually administered orally to the neonate if maternal viral load is <50 copies/ml. Otherwise triple ART should be used. Therapy should be continued for 4-6 weeks.

Infant feeding

- in the UK all women should be advised not to breast feed



Next question

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

2014 HIV in pregnancy guidelines

3 2

BHIVA

2013 Useful patient orientated leaflet

6 2

Patient.info

HIV and pregnancy

8 1

Suggest link

Report broken link

Score: **16.7%**

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A 48-year-old woman attends with a one-week history of a productive cough and shortness of breath. She has never smoked and has no past medical history. Observations are as follows: heart rate 105 beats per minute, blood pressure 125/70 mmHg, temperature 38.4ºC, respiratory rate 20 breaths per minute, oxygen saturation 92% on air.

Blood results are as follows:

Hb	122 g/L	Male: (135-180) Female: (115 - 160)
Platelets	246 * 10^9/L	(150 - 400)
WBC	14.2 * 10^9/L	(4.0 - 11.0)
Na^+	128 mmol/L	(135 - 145)
K^+	3.8 mmol/L	(3.5 - 5.0)
Urea	7.2 mmol/L	(2.0 - 7.0)
Creatinine	105 µmol/L	(55 - 120)
CRP	96 mg/L	(< 5)

Bilirubin	22 µmol/L	(3 - 17)
ALP	115 u/L	(30 - 100)
ALT	262 u/L	(3 - 40)
Î³GT	114 u/L	(8 - 60)
Albumin	32 g/L	(35 - 50)

A chest x-ray was performed:

Chest x-ray	Bilateral patchy consolidation

What treatment is indicated?

- Co-amoxiclav
- Co-trimoxazole
- Co-trimoxazole and prednisolone
- Erythromycin

Piperacillin/tazobactam

Submit answer

Reference ranges

Score: **16.7%**

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ALT	262 u/L	(3 - 40)
Î³GT	114 u/L	(8 - 60)
Albumin	32 g/L	(35 - 50)

A chest x-ray was performed:

Chest x-ray	Bilateral patchy consolidation

What treatment is indicated?

- Co-amoxiclav 13%
- Co-trimoxazole 8%
- Co-trimoxazole and prednisolone 10%
- **Erythromycin 63%**

Piperacillin/tazobactam 6%

Macrolides such as clarithromycin are used to treat *Legionella*

Important for me Less important

Erythromycin is correct. The clinical features are suggestive of *Legionella* infection. The patient has an atypical pneumonia with bilateral patchy consolidation, hyponatraemia, and deranged liver function tests, all suggestive of *Legionella* infection. Most guidelines recommend either a fluoroquinolone or a macrolide as first-line treatment for Legionella pneumonia, often with the addition of a penicillin for a synergistic effect.

Co-amoxiclav is incorrect. This antibiotic is effective for typical organisms such as *Streptococci* and *Haemophilus*.

Co-trimoxazole is incorrect. This drug can be used to treat pneumocystis pneumonia (PCP) which is a serious infection caused by the fungus *Pneumocystis jirovecii*. Although this can present similarly, it is a condition which tends to only develop in patients with weak immune systems. The patient in this clinical case is immunocompetent making PCP an unlikely diagnosis.

Co-trimoxazole and prednisolone is incorrect. This regime of drugs can be used to treat severe PCP infections characterised by a PaO2 < 8kPa.

Piperacillin/tazobactam is incorrect. This drug has a similar spectrum to co-amoxiclav with the addition of activity against *Pseudomonas*.

Discuss Improve

Next question

Legionella

Legionnaire's disease is caused by the intracellular bacterium *Legionella pneumophilia*. It typically colonizes water tanks and hence questions may hint at air-conditioning systems or foreign holidays. Person-to-person transmission is not seen

Features

- flu-like symptoms including fever (present in > 95% of patients)
- dry cough
- relative bradycardia
- confusion
- lymphopaenia
- hyponatraemia

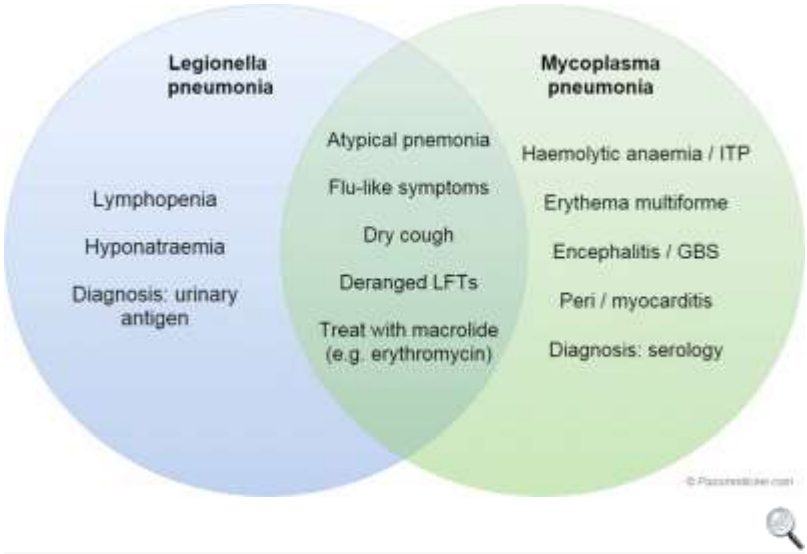
- deranged liver function tests
- pleural effusion: seen in around 30% of patients

Investigations

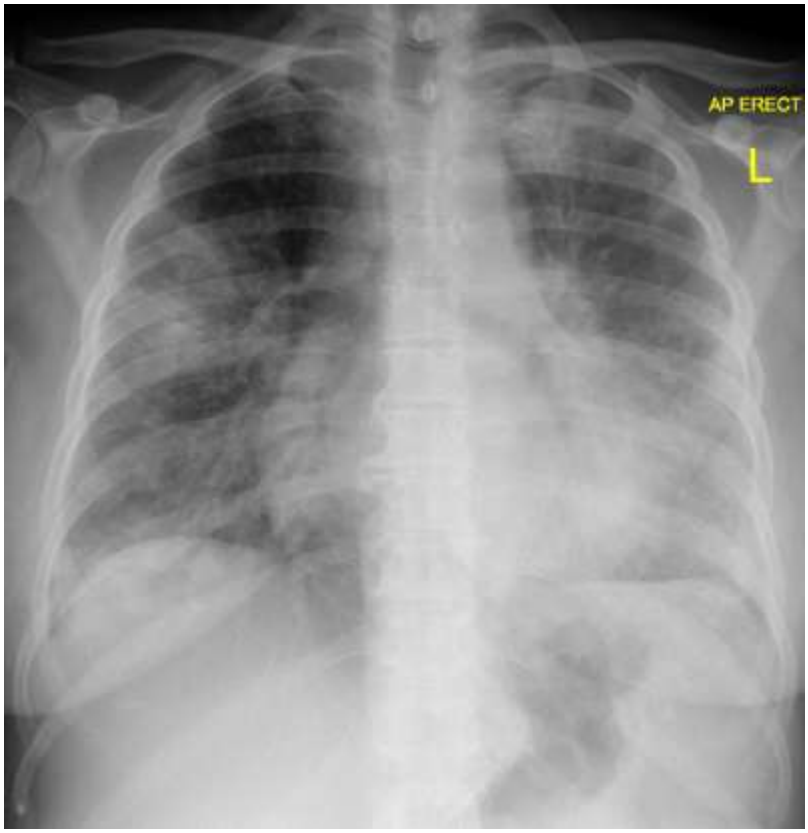
- diagnositic test of choice:urinary antigen
- chest x-ray findings are non-specific but may include:
    - a mid-to-lower zone predominance of patchy consolidation
    - pleural effusions in around 30%

Management

- treat with erythromycin/clarithromycin



Comparison of Legionella and Mycoplasma pneumonia



© Image used on license from Radiopaedia

Chest x-ray features of legionella pnuemonia are non-specific but includes a mid-to-lower zone predominance of patchy consolidation. Pleural effusions are seen in around 30%.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media



Legionella pneumophila

Osmosis - YouTube 4 0

Report broken media

Score: **16.7%**

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A 29-year-old woman is brought to the emergency department by her husband with drowsiness. Over the last day she had been complaining of headaches, nausea and fevers. About a week ago she had a period where she felt unwell with fever, headache and myalgia, but this self-resolved. She had returned from Poland 2 weeks ago where she had been on a camping holiday with friends. Her past medical history consists only of asthma, for which she takes salbutamol as required.

Examination reveals a Glasgow Coma Scale (GCS) of 14 with neck stiffness but no focal neurology. Her lungs are clear and her abdomen is soft and non-tender. There is no rash.

A lumbar puncture is performed which reveals mildly elevated white blood cells. An HIV test is negative. The patient is then started on ceftriaxone, dexamethasone and aciclovir.

The CSF culture and PCR come back negative. A sample had also been sent away to the infectious disease laboratory, which after serological testing comes back as positive for *flavivirus* antibodies.

Given the most likely diagnosis, what is the most appropriate management?

- IV amphotericin B
- IV aciclovir
- IV pyrimethamine/sulfadiazine and folinic acid
- IV quinine dihydrochloride
- Supportive management

Submit answer

Reference ranges

Score: **16.7%**

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The CSF culture and PCR come back negative. A sample had also been sent away to the infectious disease laboratory, which after serological testing comes back as positive for *flavivirus* antibodies.

Given the most likely diagnosis, what is the most appropriate management?

IV amphotericin B	3%
IV aciclovir	8%
IV pyrimethamine/sulfadiazine and folinic acid	6%
IV quinine dihydrochloride	4%
Supportive management	**78%**

Treatment for tick-borne encephalitis is supportive

Important for me Less important

A presentation of headache, neck stiffness, fever and reduced GCS is suggestive of meningoencephalitis. The question revolves around determining what is the most likely causative agent and therefore the most appropriate treatment.

The history of recent travel should raise your suspicion for a potentially uncommon aetiology. The camping history combined with a biphasic illness is characteristic for tick-borne encephalitis. Ultimately, the finding of a *flavivirus* in the CSF confirms the diagnosis. Management for this condition is supportive.

Amphotericin B may be used in the treatment of primary amoebic meningoencephalitis. This is a very rare cause of meningoencephalitis caused by *naegleria fowleri*.

Aciclovir is used in the management of herpes simplex virus (HSV) encephalitis. HSV, along with varicella-zoster virus and enteroviruses make up 90% of cases of viral encephalitis. In this patient, the travel history and biphasic illness suggest an alternate, atypical cause.

IV pyrimethamine/sulfadiazine and folinic acid are used in the management of cerebral toxoplasmosis. Toxoplasmosis is usually asymptomatic or mild in immunocompetent patients. The patient only has a past medical history of asthma and is HIV negative, therefore this is a less likely diagnosis.

IV quinine dihydrochloride may be used in severe or complicated falciparum malaria. The patient has not come from a malaria-risk country and does not have other features suggestive of malaria, such as jaundice.

Discuss | Improve

Next question >

Tick-borne encephalitis

Carried by ticks hosted by native wildlife, tick-borne encephalitis is actually caused by a virus (Flavivirus) and transmitted by the bite of an infected tick.

Once transmitted to the host, flavivirus infection manifests as a biphasic illness. During the first phase, patients may describe constitutional upset reporting headaches, myalgia, and fevers. There is typically then an asymptomatic period before the disease course progresses to phase two, which is characterized by infection by symptoms of central nervous system involvement, typically meningitis or, as the name suggests, encephalitis. The time period between phase one and two may be highly variable or absent in the case of severe infection.

Symptoms usually manifest within 7 days of infection, however, the incubation period can be up to a month. Long term neurological sequelae may persist for months - years following infection.

There are three species of flavivirus implicated in tick-borne encephalitis: European, Far Eastern and Siberian, with the Far Eastern species typically causing the most severe illness, often progressing rapidly to central nervous system involvement with no asymptomatic period, (European centre for Disease Prevention and Control quotes a mortality rate of around 35% in cases of Far Eastern flavivirus species infection).

Diagnosis is made on the basis of clinical suspicion, with patients showing characteristic symptoms and travel history confirming recent travel to affected areas. Diagnosis may be confirmed by

cerebrospinal fluid (CSF) analysis demonstrating specific IgM or IgG antibodies.

Treatment is supportive, however, the addition of doxycycline or a cephalosporin may be advised if Lyme disease is considered a differential diagnosis until confirmation via CSF sampling can be obtained.

A vaccination available and recommended to those travelling to endemic areas and planning to engage in high-risk outdoor activities, for example, hiking in rural forested areas and/or grasslands. Precautions to avoid tick bites are recommended to all travellers to endemic areas.



Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 40-year-old woman is referred to the infectious diseases team after attending her GP practice requesting advice regarding antibiotic prophylaxis for Lyme disease. The patient explains that she is planning on a 2 week long walking holiday in the Lake District National Park in Cumbria, United Kingdom and that she is concerned about the risk associated with receiving tick bites during this period. Lyme disease is a particular concern for the patient as her younger sister had suffered serious neurological complications from the condition secondary to a tick bite in the Scottish Highlands. Concern about contracting Lyme disease herself had prevented her from partaking in her hobby of cross country walking for the past 2 years but she had now decided to confront her fear by undertaking the planned trip. After undertaking research on the Internet, the patient was keen to receive prophylactic antibiotics against *Borrelia* species during her trip.

The patient's past medical history included a diagnosis of breast cancer seven years previously. Following this diagnosis, the patient had undergone a wide local excision of the tumour with postoperative chemotherapy. The patient had been given the all clear from her oncologists after five years and had been subsequently been discharged from follow-up. In addition, the patient had experienced long-standing and, at times, troubling symptoms of irritable bowel syndrome.

The patient took regular hyoscine butylbromide as treatment of her gastrointestinal symptoms. The patient stated that she was allergic to penicillin-based antibiotics. Close questioning on this topic revealed that her adverse reaction to a previous course of penicillin V had been a protracted episode of diarrhoea.

The patient lives with her husband and three teenage children. She was employed full-time as a music teacher. The patient did not smoke cigarettes and consumed approximately 10 units of alcohol per week.

What is appropriate management following the patient's request for antibiotic prophylaxis against Lyme disease during her walking holiday?

- Prescribe amoxicillin for the duration of the patient's holiday
- Prescribe amoxicillin for immediate use if she receives a tick bite
- Prescribe doxycycline for the duration of the patient's holiday continued for an additional 10 days after her return home
- Advice on the prevention and management of tick bites only
- Prescribe doxycycline for immediate use if she receives a tick bite

Submit answer

Reference ranges

Score: **16.7%**

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What is appropriate management following the patient's request for antibiotic prophylaxis against Lyme disease during her walking holiday?

Prescribe amoxicillin for the duration of the patient's holiday	2%
Prescribe amoxicillin for immediate use if she receives a tick bite	1%
Prescribe doxycycline for the duration of the patient's holiday continued for an additional 10 days after her return home	19%
Advice on the prevention and management of tick bites only	66%
Prescribe doxycycline for immediate use if she receives a tick bite	13%

There is no need for prophylactic antibiotics for Lyme disease in asymptomatic patients bitten by a tick

Important for me Less important

There is no role for antibiotic prophylaxis against Lyme disease. Simple advice on prevention of tick bites includes wearing clothing that adequately covers the patient's body and the use of insect repellent chemicals. Ticks that have attached themselves should be gently removed using tweezers to minimise the risk of rupturing the tick and potentially releasing infected material.

Individuals showing symptoms of Lyme disease - for example, erythema migrans - following potential exposure to tick bites should be treated with a 14-21 day course of doxycycline or amoxicillin immediately.

Individuals with a history of tick bite but without symptoms of Lyme disease do not normally require either treatment or serological testing. Treatment of asymptomatic individuals who have received a tick bite may be appropriate in certain situations; for example, in individuals who are immunosuppressed or who received tick bites in high-risk regions of the USA.

Duncan C, Carle G, Seaton R. Tick bite and early Lyme borreliosis. *BMJ* 2012;344:e3124.

Discuss (7) | Improve

Next question >

Lyme disease ★

Lyme disease is caused by the spirochaete *Borrelia burgdorferi* and is spread by ticks.

Early features (within 30 days)

- erythema migrans
  - 'bulls-eye' rash is typically at the site of the tick bite
  - typically develops 1-4 weeks after the initial bite but may present sooner
  - usually painless, more than 5 cm in diameter and slowlly increases in size
  - present in around 80% of patients.
- systemic features
  - headache
  - lethargy
  - fever
  - arthralgia





Later features (after 30 days)

- cardiovascular
  - heart block
  - peri/myocarditis
- neurological
  - facial nerve palsy
  - radicular pain
  - meningitis



Investigation

- NICE recommend that Lyme disease can be diagnosed clinically if erythema migrans is present
  - erythema migrans is therefore an indication to start antibiotics
- enzyme-linked immunosorbent assay (ELISA) antibodies to *Borrelia burgdorferi* are the first-line test
  - if negative and Lyme disease is still suspected in people tested within 4 weeks from symptom onset, repeat the ELISA 4-6 weeks after the first ELISA test. If still suspected in people who have had symptoms for 12 weeks or more then an immunoblot test should be done
  - if positive or equivocal then an immunoblot test for Lyme disease should be done

Management of asymptomatic tick bites

- tick bites can be a relatively common presentation to GP practices, and can cause significant anxiety
- if the tick is still present, the best way to remove it is using fine-tipped tweezers, grasping the tick as close to the skin as possible and pulling upwards firmly. The area should be washed following.
- NICE guidance does not recommend routine antibiotic treatment to patients who've suffered a tick bite

Management of suspected/confirmed Lyme disease

- doxycycline if early disease. Amoxicillin is an alternative if doxycycline is contraindicated (e.g. pregnancy)

    - people with erythema migrans should be commenced on antibiotic without the need for further tests
- ceftriaxone if disseminated disease
- Jarisch-Herxheimer reaction is sometimes seen after initiating therapy: fever, rash, tachycardia after first dose of antibiotic (more commonly seen in syphilis, another spirochaetal disease)

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Next question

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High-yield textbook

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Links

Clinical Knowledge Summaries 11 13

Lyme disease: management

NICE 11 10

2018 Lyme disease guidelines

Dermnet NZ 12 5

Lyme disease

Suggest link Report broken link

Media



Lyme disease

Osmosis - YouTube 4 1

Report broken media

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A 35-year-old male attends clinic with a new diagnosis of chronic hepatitis B infection. He gives a history of unprotected sex with multiple sexual partners in the two years. His blood tests show him to be HIV, HCV antibody and HBeAg negative. The most recent liver function tests are shown below. On examination, he has no evidence of decompensated liver disease.

Bilirubin	18µmol/L
ALT	52iu/L
AST	38iu/L
ALP	190iu/L
Albumin	30g/L
Protein	93g/L
HBV viral load	2,000IU/mL

Which one of the following treatment options should be considered first line?

- Peginterferon alpha 2a and entecavir
- Entecavir
- Peginterferon alpha 2a
- Tenofovir disoproxil
- Entecavir and tenofovir disoproxil

Submit answer

Reference ranges

Score: **16.7%**

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HBV viral load	2,000IU/mL

Which one of the following treatment options should be considered first line?

Peginterferon alpha 2a and entecavir	23%
Entecavir	11%
Peginterferon alpha 2a	36%
Tenofovir disoproxil	13%
Entecavir and tenofovir disoproxil	18%

Peginterferon alpha 2a is the first line treatment for adults with HBeAg-negative chronic hepatitis B with compensated liver disease. Tenofovir disoproxil or entecavir are second line treatment in those who have detectable HBV DNA after treatment with peginterferon alpha 2a.

Hepatitis B (chronic): diagnosis and management of chronic hepatitis B in children, young people and adults (2013)

Discuss (6) | Improve

Next question >

Hepatitis B ★

Hepatitis B is a double-stranded DNA hepadnavirus and is spread through exposure to infected blood or body fluids, including vertical transmission from mother to child. The incubation period is 6-20 weeks.

The features of hepatitis B include fever, jaundice and elevated liver transaminases.

Complications of hepatitis B infection

- chronic hepatitis (5-10%). 'Ground-glass' hepatocytes may be seen on light microscopy
- fulminant liver failure (1%)
- hepatocellular carcinoma
- glomerulonephritis
- polyarteritis nodosa
- cryoglobulinaemia

Immunisation against hepatitis B (please see the Greenbook link for more details)

- children born in the UK are now vaccinated as part of the routine immunisation schedule. This is given at 2, 3 and 4 months of age
- at risk groups who should be vaccinated include: healthcare workers, intravenous drug users, sex workers, close family contacts of an individual with hepatitis B, individuals receiving blood transfusions regularly, chronic kidney disease patients who may soon require renal replacement therapy, prisoners, chronic liver disease patients
- contains HBsAg adsorbed onto aluminium hydroxide adjuvant and is prepared from yeast cells using recombinant DNA technology
- around 10-15% of adults fail to respond or respond poorly to 3 doses of the vaccine. Risk factors include age over 40 years, obesity, smoking, alcohol excess and immunosuppression
- testing for anti-HBs is only recommended for those at risk of occupational exposure (i.e. Healthcare workers) and patients with chronic kidney disease. In these patients anti-HBs levels should be checked 1-4 months after primary immunisation
- the table below shows how to interpret anti-HBs levels:

Anti-HBs level (mIU/ml)	**Response**
> 100	Indicates adequate response, no further testing required. Should still receive booster at 5 years
10 - 100	Suboptimal response - one additional vaccine dose should be given. If immunocompetent no further testing is required
< 10	Non-responder. Test for current or past infection. Give further vaccine course (i.e. 3 doses again) with testing following. If still fails to respond then HBIG

Anti-HBs level (mIU/ml)	Response
	would be required for protection if exposed to the virus

Management of hepatitis B

- pegylated interferon-alpha used to be the only treatment available. It reduces viral replication in up to 30% of chronic carriers. A better response is predicted by being female, < 50 years old, low HBV DNA levels, non-Asian, HIV negative, high degree of inflammation on liver biopsy
- whilst NICE still advocate the use of pegylated interferon firstl-line other antiviral medications are increasingly used with an aim to suppress viral replication (not in a dissimilar way to treating HIV patients)
- examples include tenofovir, entecavir and telbivudine (a synthetic thymidine nucleoside analogue)



Next question

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Extended textbook

Links

NICE

7 7

2013 Chronic hepatitis B guidelines

Green Book

8 5

Hepatitis B guidelines

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Media



Viral hepatitis

Osmosis - YouTube 5 0



Anti-hepatitis medications

Osmosis - YouTube 1 0



Hepatitis

Podmedics - YouTube 1 1

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A 43-year-old gentleman presents with bloody diarrhoea. He is HIV positive and has a CD4 count of 150 cells/µL.

He had been on a trip to Zimbabwe 10 months previously. Over the past 2 weeks, he had symptoms of anorexia and mild abdominal discomfort. These had progressed on to severe abdominal cramps and bloody diarrhoea.

He has a temperature of 37.8ºC, a blood pressure of 100/60 and a pulse of 111/min. On examination, he looks unwell, is emaciated and has generalised tenderness on examination of his abdomen. There is no guarding or peritonitis.

What is the most likely causative organism?

- *Giardia lamblia* ✗
- *Toxoplasma gondii* ✗
- *Entamoeba histolytica* ✗
- *Cytomegalovirus* ✗
- *Strongyloides stercoralis* ✗

Submit answer

Reference ranges

Score: **16.7%**

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What is the most likely causative organism?

Giardia lamblia	10%
Toxoplasma gondii	5%
Entamoeba histolytica	41%
Cytomegalovirus	40%
Strongyloides stercoralis	4%

Entamoeba histolytica is the only pathogen here which typically causes bloody diarrhoea in an immunocompromised individual and hence is the correct answer. Giardia typically causes steatorrhoea, *Toxoplasma gondii* is more likely to cause a central nervous system infection, and while CMV can cause diarrhoea it is rarely a presenting feature of the infection. While *Strongyloides stercoralis* can cause diarrhoea, it is usually watery rather than bloody.

Discuss (12) | Improve

Next question >

Amoebiasis

Amoebiasis is caused by *Entamoeba histolytica* (an amoeboid protozoan) and spread by the faecal-oral route. It is estimated that 10% of the world's population is chronically infected.

Infection can be asymptomatic, cause mild diarrhoea or severe amoebic dysentery. Amoebiasis also causes liver and colonic abscesses.

Amoebic dysentery

- profuse, bloody diarrhoea
- there may be a long incubation period
- stool microscopy may show trophozoites if examined within 15 minutes or kept warm (known as a 'hot stool')
- treatment
  - oral metronidazole
  - a 'luminal agent' (to eliminate intraluminal cysts) is recommended usually as well e.g. diloxanide furoate

Amoebic liver abscess

- usually a single mass in the right lobe (may be multiple). The contents are often described as 'anchovy sauce'
- features
  - fever
  - right upper quadrant pain
  - systemic symptoms e.g. malaise
  - hepatomegaly
- investigations
  - ultrasound
  - serology is positive in > 95%
- management
  - oral metronidazole
  - a 'luminal agent' (to eliminate intraluminal cysts) is recommended usually as well e.g. diloxanide furoate

AI + Q 123

Next question >

Textbooks

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Links

Patient.info

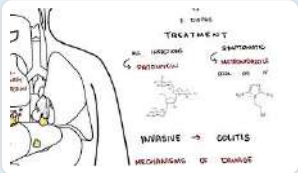
13 6

Amoebiasis review

Suggest link

Report broken link

Media



Amebiasis - entamoeba histolytica symptoms, pathophysiology, diagnosis, treatment

Armando Hasudungan - YouTube

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A 25-year-old woman returns from a holiday in Thailand with diarrhoea. She has no past medical history and takes no regular tablets apart from the oral contraceptive pill. She has no allergies apart from peanuts. She has a been suffering from diarrhoea for three days with mild abdominal cramps which started five days after returning from her trip. There has been a small amount of blood in her stool. Her observations are within normal range. She has no postural symptoms. Stool samples are requested and sent to the microbiology lab. What is the most appropriate treatment?

- Ciprofloxacin ×
- Tetracycline ×
- Oral rehydration solution ×
- Metronidazole ×
- Co-amoxiclav ×

Submit answer

Reference ranges

Score: **16.7%**

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Ciprofloxacin	34%
Tetracycline	2%
Oral rehydration solution	34%
Metronidazole	29%
Co-amoxiclav	1%

The correct answer is oral rehydration solution. Empirical antibiotics are only indicated if systemically unwell, immunosuppressed or if elderly, even if the patient has bloody diarrhoea. CIprofloxacin is used to treat Salmonella, Shigella and Campylobacter, tetracycline can be used for cholera, metronidazole in parasitic infections or other bowel problems, and finally, co-amoxiclav is seldom used in enteric infections.

Discuss (6) | Improve

Next question

Gastroenteritis: causes

Gastroenteritis may either occur whilst at home or whilst travelling abroad (travellers' diarrhoea)

Travellers' diarrhoea may be defined as at least 3 loose to watery stools in 24 hours with or without one of more of abdominal cramps, fever, nausea, vomiting or blood in the stool. The most common cause is *Escherichia coli*.

Another pattern of illness is 'acute food poisoning'. This describes the sudden onset of nausea, vomiting and diarrhoea after the ingestion of a toxin. Acute food poisoning is typically caused by

Staphylococcus aureus, *Bacillus cereus* or *Clostridium perfringens*.

Stereotypical histories

Infection	Typical presentation
Escherichia coli	Common amongst travellers Watery stools Abdominal cramps and nausea
Giardiasis	Prolonged, non-bloody diarrhoea
Cholera	Profuse, watery diarrhoea Severe dehydration resulting in weight loss Not common amongst travellers
Shigella	Bloody diarrhoea Vomiting and abdominal pain
Staphylococcus aureus	Severe vomiting Short incubation period
Campylobacter	A flu-like prodrome is usually followed by crampy abdominal pains, fever and diarrhoea which may be bloody May mimic appendicitis Complications include Guillain-Barre syndrome
Bacillus cereus	Two types of illness are seen • vomiting within 6 hours, stereotypically due to rice • diarrhoeal illness occurring after 6 hours
Amoebiasis	Gradual onset bloody diarrhoea, abdominal pain and tenderness which may last for several weeks

Incubation period

- 1-6 hrs: *Staphylococcus aureus*, *Bacillus cereus**
- 12-48 hrs: *Salmonella*, *Escherichia coli*
- 48-72 hrs: *Shigella*, *Campylobacter*
- > 7 days: Giardiasis, Amoebiasis

*vomiting subtype, the diarrhoeal illness has an incubation period of 6-14 hours

AI + Q 123

Next question >

Textbooks

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Extended textbook

Links

Royal College of Physicians 16 13

2011 Diarrhoeal disease review

Suggest link Report broken link

Media



Shigella

ATP - YouTube 5 0



Salmonella

ATP - YouTube 5 0

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Score: **16.7%**

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A 25-year-old man presents to gastroenterology with a 6-month history of unexplained chronic diarrhoea and unexplained chronic weight loss. An HIV test is sent as part of his work-up and returns positive. His CD4 count is 150. He is started on anti-retroviral therapy.

Round oocysts measuring between 4 and 6 µm are seen during microscopy of the patient's stool sample. They stain red using the modified acid-fast stain. A diagnosis of Cryptosporidium is made. The first-line treatment of nitazoxanide is not available.

What other medication could be a potential treatment option for this patient?

- Ciprofloxacin
- Metronidazole
- Nitrofurantoin
- Prednisolone
- Rifaximin

Submit answer

Reference ranges

Score: **16.7%**

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What other medication could be a potential treatment option for this patient?

Ciprofloxacin	13%
Metronidazole	38%
Nitrofurantoin	3%
Prednisolone	2%
Rifaximin	**44%**

Rifaxamin is licensed for the treatment of cryptosporidiosis

Important for me Less important

The oocysts described on stool microscopy are typical of *Cryptosporidium*. *Cyclospora* and *Isospora* oocysts will also stain with the modified acid-fast stain. The oocysts of *Cyclospora* look similar to those of *Cryptosporidium* but are larger at 8-10 µm. *Isospora* oocysts are oval shaped and are larger again at 10-20 x 20-30 µm.

Cryptosporidiosis is a protozoal infection that generally causes mild, watery diarrhoea in immune-competent patients, although symptoms can persist for several weeks. It can cause chronic diarrhoea in immune suppressed patients, especially those with HIV infection. Treatment is not always required as the symptoms often improve once the immune system starts to recover. If treatment is indicated the first line option is nitazoxanide, followed by rifaximin.

Ciprofloxacin can be used for some causes of infectious diarrhoea, such as *Campylobacter* and *Salmonella* but does not treat *Cryptosporidium*.

Metronidazole is effective against anaerobic bacteria and some protozoa (including *Giardia*) but is not active against *Cryptosporidium*.

Nitrofurantoin is mainly used for the treatment of lower urinary tract infections due to Gram negative rods and will not treat *Cryptosporidium*.

Prednisolone may worsen the patient's symptoms by increasing his level of immune suppression so would not be a good treatment option in this case.

Discuss (4) | Improve

Next question >

Cryptosporidiosis

Cryptosporidiosis is the commonest protozoal cause of diarrhoea in the UK. Two species, *Cryptosporidium hominis* and *Cryptosporidium parvum* account for the majority cases.

Cryptosporidiosis is more common in immunocompromised patients (e.g. HIV) and young children.

Features

- watery diarrhoea
- abdominal cramps
- fever
- in immunocompromised patients, the entire gastrointestinal tract may be affected resulting in complications such as sclerosing cholangitis and pancreatitis

Diagnosis

- stool: modified Ziehl-Neelsen stain (acid-fast stain) of the stool may reveal the characteristic red cysts of Cryptosporidium

Management

- is largely supportive for immunocompetent patients
- if the patient has HIV and is not on antiretroviral therapy then this should be started and often will be enough to resolve the infection
- nitazoxanide may be used for immunocompromised patients
- rifaximin is also sometimes used for immunocompromised patients/patients with severe disease

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 38-year-old man from Nigeria presents to a gastroenterology clinic. He is a businessman, working for an iron and steel trading company and makes regular trips to Swaziland to visit mining executives.

For the previous four months, he has been troubled by diarrhoea, up to 8 episodes a day, with abdominal bloating and cramping. There is no blood visible in the stool. He has previously been treated for tuberculosis as a child.

A stool sample is sent to the lab, and Modified Ziehl-Neelson stain reveals multiple red staining round objects measuring 5 microns in diameter.

What will be the most appropriate treatment?

- Highly active anti-retroviral therapy
- Co-trimoxazole
- Rifampicin, isoniazid, ethambutol, pyrazinamide
- Metronidazole
- Ciprofloxacin

Submit answer

Reference ranges

Score: **16.7%**

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A stool sample is sent to the lab, and Modified Ziehl-Neelson stain reveals multiple red staining round objects measuring 5 microns in diameter.

What will be the most appropriate treatment?

Highly active anti-retroviral therapy	39%
Co-trimoxazole	14%
Rifampicin, isoniazid, ethambutol, pyrazinamide	15%
Metronidazole	24%
Ciprofloxacin	7%

The diagnosis is cryptosporidium infection. It is the most common cause of chronic diarrhoea in HIV patients. Diagnosis is made by visualisation with modified Zeihl-Neelson stain of stool. You would very rarely expect to see *Mycobacterium tuberculosis* on a faecal sample.

Discuss (11) | Improve

Next question

Cryptosporidiosis

Cryptosporidiosis is the commonest protozoal cause of diarrhoea in the UK. Two species, *Cryptosporidium hominis* and *Cryptosporidium parvum* account for the majority cases.

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Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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Mrs Stevens is a 60-year-old lady with pulmonary fibrosis secondary to dermatomyositis. She returns to your clinic seven weeks after being commenced on high dose steroids due to disease progression. During the review Mrs Stevens describes increasing shortness of breath, which is worse when she exerts herself. She has not noticed any fever and has not lost any weight. She has a chronic cough productive of white sputum. This remains stable.

On examination she looks comfortable at rest and auscultation of her chest is surprisingly clear. At rest her oxygen Sats are 97%. You ask her to walk to the end of the corridor and back. You notice that on returning her Sats have fallen to 82%. You request an urgent chest radiograph, which is reported as showing bilateral patchy infiltrates.

What is the most appropriate management option?

- Fluconazole
- Ambisome
- Co-trimoxazole
- Ribavirin
- Stop steroids

Submit answer

Reference ranges

Score: **16.7%**

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What is the most appropriate management option?

Fluconazole	4%
Ambisome	4%
Co-trimoxazole	87%
Ribavirin	2%
Stop steroids	4%

Pneumocystis jiroveci penumonia is treated with co-trimoxazole, which is a mix of trimethoprim and sulfamethoxazole

Important for me Less important

The patient has PCP pneumonia, caused by *Pneumocystis jirovecii* - a yeast-like fungus which is an opportunistic pathogen. The treatment for this is co-trimoxazole.

All patients with dermatomyositis or polymyositis with pulmonary fibrosis who are treated with glucocorticoids are advised to receive PCP prophylaxis with co-trimoxazole. Stopping steroids would not be appropriate in this scenario.

It is important to note that not all patients taking glucocorticoids for rheumatological conditions require PCP prophylaxis. However, there is evidence that in non-HIV cohorts, the greatest risk for developing PCP is glucocorticoid use and deficiencies in innate and acquired immune components. Therefore, although there are currently no formal guidelines it is recommended that if an individual satisfies both these criteria in rheumatological conditions, they should be

considered for PCP prophylaxis.

For example the following all should be considered for prophylaxis:

- A patient on the equivalent of 20 mg prednisolone or greater for one month plus the concomitant use of another immunosuppressive drug
- Dermatomyositis on high dose prednisolone for greater than one month and pulmonary fibrosis
- Allogenic or autogenic stem cell transplant or solid organ transplantation

Further reading can be found at:
http://www.pneumon.org/assets/files/844/file483317.pdf

Discuss (3) | Improve

Next question >

HIV: *Pneumocystis jiroveci* pneumonia

Whilst the organism *Pneumocystis carinii* is now referred to as *Pneumocystis jiroveci*, the term *Pneumocystis carinii* pneumonia (PCP) is still in common use

- *Pneumocystis jiroveci* is an unicellular eukaryote, generally classified as a fungus but some authorities consider it a protozoa
- PCP is the most common opportunistic infection in AIDS
- all patients with a CD4 count < 200/mm^3 should receive PCP prophylaxis

Features

- dyspnoea
- dry cough
- fever
- very few chest signs

Pneumothorax is a common complication of PCP.

Extrapulmonary manifestations are rare (1-2% of cases), may cause

- hepatosplenomegaly
- lymphadenopathy
- choroid lesions

Investigation

- CXR: typically shows bilateral interstitial pulmonary infiltrates but can present with other x-ray findings e.g. lobar consolidation. May be normal
- exercise-induced desaturation
- sputum often fails to show PCP, bronchoalveolar lavage (BAL) often needed to demonstrate PCP (silver stain shows characteristic cysts)





Management

- co-trimoxazole
- IV pentamidine in severe cases
- aerosolized pentamidine is an alternative treatment for *Pneumocystis jiroveci* pneumonia but is less effective with a risk of pneumothorax
- steroids if hypoxic (if pO2 < 9.3kPa then steroids reduce risk of respiratory failure by 50% and death by a third)

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

BNF

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Pneumocystis pneumonia

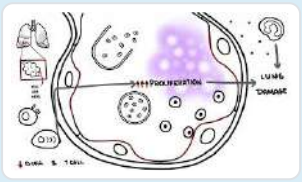
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Pneumocystis Jirovecii Pneumonia review

Suggest link Report broken link

Media



Pneumocystis jirovecii - Pneumocystis pneumonia

Armando Hasudungan - YouTube

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Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

2 1

Report broken media

Score: **16.7%**

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A 43-year-old man presents to his local sexual health clinic with an indurated well-circumscribed painless ulcer on the glans of his penis. Further examination demonstrates inguinal lymphadenopathy. He has had unprotected vaginal intercourse with a causal female partner within the last 14 days. He identifies as heterosexual, was born in London. He consents to a full sexual health screening and is prescribed a 14-day course of doxycycline as he reports a previous urticarial rash associated with penicillin.

The following day, the patient returns to the clinic with a high fever and widespread rash. He is concerned that he may have an allergy to doxycycline and would like advice regarding an alternate treatment. He reports no respiratory distress, tongue swelling or headache and consents to a set of monitoring blood tests, the results of which are within normal limits.

Given the likely diagnosis, which of the following would be the most appropriate course of action?

- Advise the patient that his rash is likely due to sun exposure with concomitant doxycycline use
- Give a cephalosporin based treatment regime instead
- Reassure the patient that this reaction with treatment of syphilis and advice paracetamol with good oral hydration
- Admit immediately to hospital
- Take a p24 antigen test and counsel the patient about possible HIV seroconversion

Submit answer

Reference ranges

Score: **16.7%**

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Given the likely diagnosis, which of the following would be the most appropriate course of action?

Advise the patient that his rash is likely due to sun exposure with concomitant doxycycline use	6%
Give a cephalosporin based treatment regime instead	3%
Reassure the patient that this reaction with treatment of syphilis and advice paracetamol with good oral hydration	79%
Admit immediately to hospital	3%
Take a p24 antigen test and counsel the patient about possible HIV seroconversion	8%

The Jarisch-Herxheimer reaction is a known phenomenon following syphilis treatment that does not require any specific treatment or investigations other antipyretics

Important for me Less important

This patient has syphilis and is experiencing a Jarisch-Herxheimer reaction, the mechanism of which remains poorly understood, but is felt to be related to the immune response triggered by the killed organisms.

Doxycycline is the treatment of choice in patients with a penicillin allergy and, given the reported allergy described by the patient, a cephalosporin would not be advised.

The Jarisch-Herxheimer reaction is usually self-limiting and best managed conservatively with antipyretics and good hydration unless there is evidence of organ compromise. Hospital admission

is unnecessary unless there is evidence of organ compromise.

HIV seroconversion is possible given the recent unprotected intercourse, but this patient would be considered low risk given his exposure and a Jarisch-Herxheimer reaction is much more likely.

Discuss (3) | Improve

Next question ›

Syphilis: management ★

Management

- intramuscular benzathine penicillin is the first-line management
- alternatives: doxycycline
- nontreponemal (rapid plasma reagin [RPR] or Venereal Disease Research Laboratory [VDRL]) titres should be monitored after treatment to assess the response
  - a fourfold decline in titres (e.g. 1:16 → 1:4 or 1:32 → 1:8)is often considered an adequate response to treatment
- the Jarisch-Herxheimer reaction is sometimes seen following treatment
  - fever, rash, tachycardia after the first dose of antibiotic
  - in contrast to anaphylaxis, there is no wheeze or hypotension
  - it is thought to be due to the release of endotoxins following bacterial death and typically occurs within a few hours of treatment
  - no treatment is needed other than antipyretics if required

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

British Association for Sexual Health and HIV

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UK National Guidelines on the Management of Syphilis 2015

Suggest link

Report broken link

Media



Syphilis

Osmosis - YouTube 1 1

Report broken media

Score: **16.7%**

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A 57-year-old man who just returned from a cruise to the Bahamas comes to the emergency department with fever, non-productive cough and shortness of breath for the past 2 days. He has also had a headache, abdominal pain and diarrhoea since yesterday. The patient has a history of type 2 diabetes mellitus, and a smoking history of 20 pack-years. He does not use alcohol or illicit drugs. His temperature is 39.2ºC, blood pressure is 110/65 mmHg, pulse is 80/min and respirations are 18/min. Pulse oximetry shows 97% on room air.

Lung examination demonstrates crackles bilaterally. The abdomen is soft and non-tender. The patient seems confused at times during the examination. His serum glucose is normal; serum sodium is 128 mmol/l. Chest x-ray demonstrates bilateral interstitial infiltrates. He is admitted to the ward.

Which of the following antibiotics should be given to this patient?

- Co-amoxiclav
- Ceftriaxone
- Clindamycin
- Levofloxacin
- Vancomycin

Submit answer

Reference ranges

Score: **16.7%**

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A 57-year-old man who just returned from a cruise to the Bahamas comes to the emergency department with fever, non-productive cough and shortness of breath for the past 2 days. He has also had a headache, abdominal pain and diarrhoea since yesterday. The patient has a history of type 2 diabetes mellitus, and a smoking history of 20 pack-years. He does not use alcohol or illicit drugs. His temperature is 39.2ºC, blood pressure is 110/65 mmHg, pulse is 80/min and respirations are 18/min. Pulse oximetry shows 97% on room air.

Lung examination demonstrates crackles bilaterally. The abdomen is soft and non-tender. The patient seems confused at times during the examination. His serum glucose is normal; serum sodium is 128 mmol/l. Chest x-ray demonstrates bilateral interstitial infiltrates. He is admitted to the ward.

Which of the following antibiotics should be given to this patient?

Co-amoxiclav	8%
Ceftriaxone	10%
Clindamycin	26%
Levofloxacin	55%
Vancomycin	1%

This patient has bilateral lung infiltrates, confusion and diarrhoea following travel. Legionella pneumonia (Legionnaires' disease) must be considered. Travel-associated Legionnaires' disease due to contaminated water supplies is a well-documented condition linked to cruise ships and hotels. *Legionella pneumophila* is a Gram-negative bacilli that stains poorly as it is primarily intracellular; hence Gram stain showing many neutrophils but no organisms is characteristic.

Legionnaires' disease is treated with antibiotics that achieve high intracellular concentration, particularly macrolides or fluoroquinolones (such as levofloxacin). The latter is favoured when the illness is severe enough to warrant admission.

Choice 1: Co-amoxiclav is typically used in some cases of CAP, but not Legionnaires' disease. It has activity against beta-lactamase-producing *Haemophilus* and *Moraxella* species.

Choice 2: Ceftriaxone has good activity against pneumococcus as well as *Haemophilus* and *Moraxella*. It does not have activity against *Legionella* or other causes of atypical pneumonia.

Choice 3: Clindamycin is used primarily to treat infections with anaerobic organisms; *Legionella* has aerobic activity.

Choice 5: Vancomycin has no activity against *Legionella* or most other causes of atypical pneumonia.

Discuss (4) | Improve

Next question >

Legionella

Legionnaire's disease is caused by the intracellular bacterium *Legionella pneumophilia*. It typically colonizes water tanks and hence questions may hint at air-conditioning systems or foreign holidays. Person-to-person transmission is not seen

Features

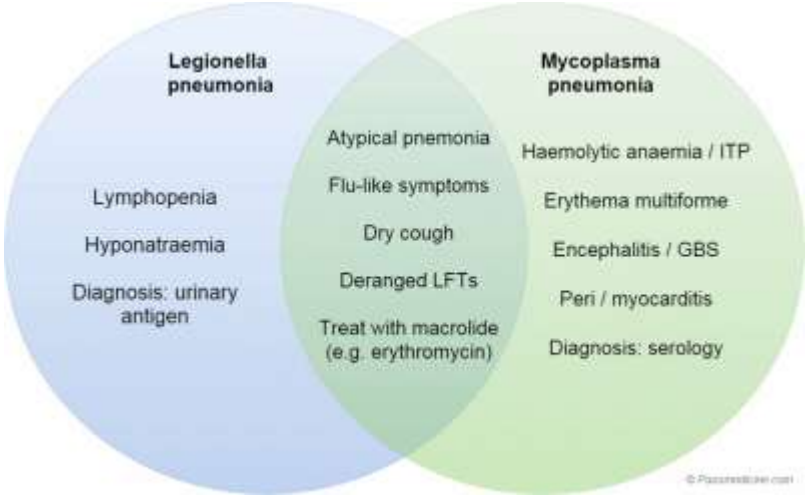
- flu-like symptoms including fever (present in > 95% of patients)
- dry cough
- relative bradycardia
- confusion
- lymphopaenia
- hyponatraemia
- deranged liver function tests
- pleural effusion: seen in around 30% of patients

Investigations

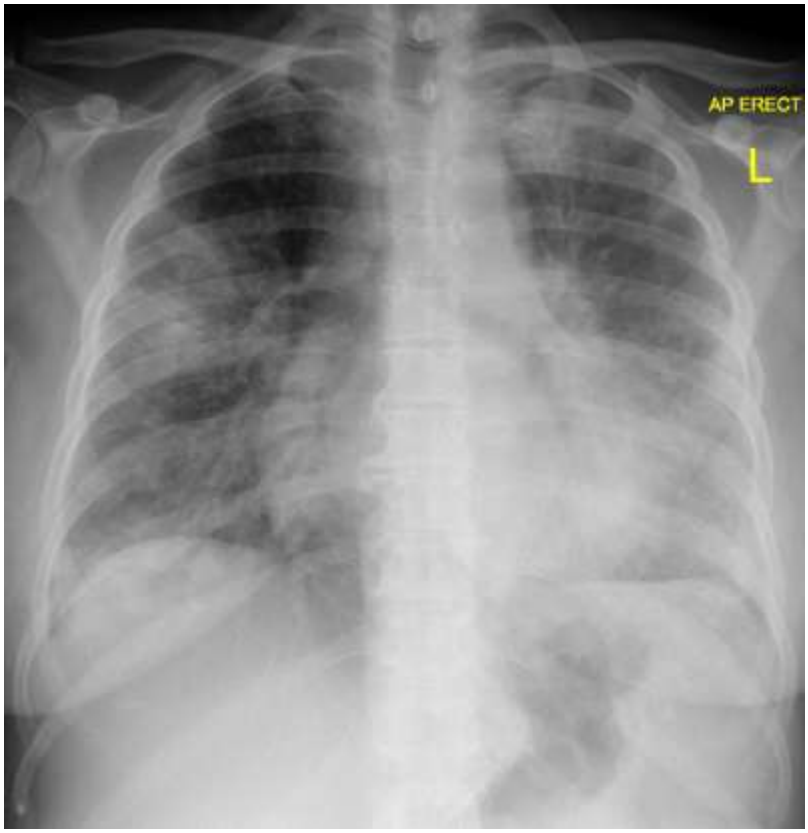
- diagnositic test of choice:urinary antigen
- chest x-ray findings are non-specific but may include:
  - a mid-to-lower zone predominance of patchy consolidation
  - pleural effusions in around 30%

Management

- treat with erythromycin/clarithromycin



Comparison of Legionella and Mycoplasma pneumonia



© Image used on license from Radiopaedia

Chest x-ray features of legionella pnuemonia are non-specific but includes a mid-to-lower zone predominance of patchy consolidation. Pleural effusions are seen in around 30%.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media



Legionella pneumophila

Osmosis - YouTube 4 0

Report broken media

Score: **16.7%**

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A 64-year-old lady with a background of Alzheimer's dementia presents with a 3 day history of increased confusion, lower abdominal pain and foul smelling urine. Blood tests on admission show:

Hb	119 g/l	Na^+	136 mmol/l
Platelets	425 * 10^9/l	K^+	3.7 mmol/l
WBC	15.9 * 10^9/l	Urea	7.2 mmol/l
Neuts	13 * 10^9/l	Creatinine	78 µmol/l
Lymphs	2 * 10^9/l	CRP	140 mg/l
Eosin	0.02 * 10^9/l		

She is started on treatment for a urinary tract infection. 2 days later her blood tests show the following:

Hb	115 g/l	Na^+	141 mmol/l
Platelets	360 * 10^9/l	K^+	5.2 mmol/l
WBC	10.2* 10^9/l	Urea	8 mmol/l
Neuts	7.5 * 10^9/l	Creatinine	105 µmol/l
Lymphs	1.5 * 10^9/l	CRP	43 mg/l
Eosin	0.001* 10^9/l		

Which antibiotic was she likely to have been treated with?

- Trimethoprim
- Erythromycin
- Co-amoxiclav
- Nitrofurantoin
- Ciprofloxacin

Submit answer

Reference ranges

Score: **16.7%**

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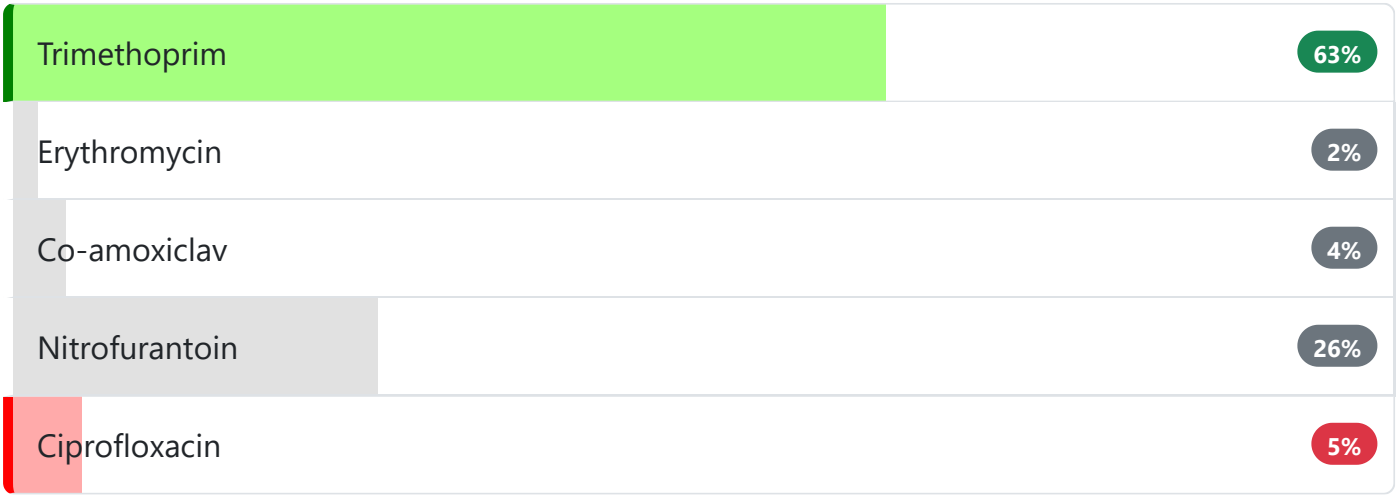
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Hb	119 g/l	Na^+	136 mmol/l
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Eosin	0.001* 10^9/l		

Which antibiotic was she likely to have been treated with?



The correct answer is **Trimethoprim**. This is evidenced by the rise in serum potassium from 3.7 mmol/L to 5.2 mmol/L over two days of treatment. Trimethoprim is known to cause hyperkalaemia

through its action on the distal tubule of the kidney where it blocks the sodium channel ENaC, leading to reduced potassium excretion. This effect is particularly notable in elderly patients and those with reduced renal function. The patient's other blood results show an appropriate response to antibiotic therapy with falling WBC and CRP.

Erythromycin is not a first-line treatment for urinary tract infections in the UK according to NICE guidelines. While it can cause various side effects, significant changes in potassium levels are not typically associated with its use. Additionally, it has poor urinary penetration making it an unsuitable choice for UTIs.

Co-amoxiclav, while commonly used for UTIs, does not typically cause hyperkalaemia. The blood results show improving inflammatory markers which could be consistent with co-amoxiclav use, but the marked rise in potassium would not be expected with this antibiotic.

Nitrofurantoin is a common first-line treatment for uncomplicated UTIs, but it would be contraindicated in this patient due to her reduced renal function (eGFR likely <45 ml/min based on her age and creatinine). Furthermore, nitrofurantoin does not typically cause electrolyte disturbances.

Ciprofloxacin, while effective against urinary pathogens, does not cause significant changes in potassium levels. While it could effectively treat this infection, the characteristic rise in potassium seen in this case would not be expected with ciprofloxacin therapy.

Discuss (5) | Improve

Next question >

Trimethoprim ★

Trimethoprim is an antibiotic, mainly used in the management of urinary tract infections.

Mechanism of action

- interferes with DNA synthesis by inhibiting dihydrofolate reductase
  - may, therefore, interact with methotrexate, which also inhibits dihydrofolate reductase

Adverse effects

- myelosuppression
- transient rise in creatinine: trimethoprim competitively inhibits the tubular secretion of creatinine resulting in a temporary increase which reverses upon stopping the drug
  - trimethoprim blocks the ENaC channel in the distal nephron, causing a hyperkalaemic distal RTA (type 4). It also inhibits creatinine secretion, often leading to an increase in creatinine by around 40 points (but not necessarily causing AKI)

Use in pregnancy

- the BNF advises that there is a: '*Teratogenic risk in first trimester (folate antagonist). Manufacturers advise avoid during pregnancy.*'

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 19-year-old man returning from Zimbabwe is admitted to the emergency department. He arrived back in the UK one week ago but for the last three days has complained of a severe headache and rigors. He has no significant past medical history. He smokes 10 cigarettes per day and does not drink alcohol.

On examination, the patient is sweaty and appears anxious. Pulse rate is 108/min, blood pressure 91/58 mmHg, temperature 39ºC and the respiratory rate 22/min. His chest is clear and heart sounds normal. A brief neurological examination is normal. An urgent blood film reveals malarial parasites. Which of the following would suggest severe malaria infection?

- Blood sugar > 14 mmol/l
- Bicarbonate < 14mmol/l
- CRP > 250 mg/l
- Temperature > 38.5ºC
- Infection despite use of appropriate antimalarial prophylaxis

Submit answer

Reference ranges

Score: **16.7%**

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On examination, the patient is sweaty and appears anxious. Pulse rate is 108/min, blood pressure 91/58 mmHg, temperature 39ºC and the respiratory rate 22/min. His chest is clear and heart sounds normal. A brief neurological examination is normal. An urgent blood film reveals malarial parasites. Which of the following would suggest severe malaria infection?

Blood sugar > 14 mmol/l	6%
Bicarbonate < 14mmol/l	72%
CRP > 250 mg/l	3%
Temperature > 38.5ºC	12%
Infection despite use of appropriate antimalarial prophylaxis	7%

The World Health Organisation states that the early recognition and treatment of severe malaria is vital to prevent rapid deterioration in the patient's condition and the onset of life-threatening complications.

Complications indicating severe malaria include:

- hypoglycaemia
- acidosis
- coma
- convulsions
- severe anaemia
- pulmonary oedema

Both coma and convulsions may represent cerebral malaria. In addition the parasite involved and percentage parasitaemia is important in the prognosis and management of malaria

Malaria: Falciparum ★

Falciparum malaria is the commonest, and most severe, type of malaria.

Clinical features

Falciparum malaria typically presents with symptoms suggestive of a flu-like illness. The classical triad of symptoms includes paroxysms of fever, chills, and sweating. These symptoms may occur every 48 hours corresponding to the erythrocytic cycle of the *Plasmodium falciparum* parasite. Fever is often high, intermittent, and may be accompanied by rigors. Initial manifestations can be non-specific and include malaise, headache, and myalgia, which might be mistaken for a viral syndrome.

General features of falciparum malaria

- fever: The hallmark of malaria, typically cyclical, often accompanied by sweating and sometimes rigors.
- gastrointestinal:
  - anorexia, nausea, vomiting, and abdominal pain are frequent
  - diarrhoea can occasionally be a feature, more commonly in children
  - patients may exhibit mild jaundice and occasional pruritus
- respiratory: Cough and, in some cases, mild tachypnoea may occur, though primary respiratory complications are rare in non-severe cases.
- musculoskeletal: Generalised body aches and joint pains are common.
- neurological:
  - headache is prominent and often severe.
  - dizziness and sleep disturbances may also be observed.
- cardiovascular: Tachycardia may be evident, and although hypotension is more typical of severe malaria, it can occasionally be seen in non-severe cases as well.
- haematological:
  - thrombocytopaenia is the most significant haematological finding and can occur in the absence of severe disease.
  - mild anaemia may also be present.
- renal: While acute kidney injury is associated with severe malaria, non-severe cases might show mild to moderate increases in creatinine or blood urea nitrogen levels.

Feature of severe falciparum malaria

- schizonts on a blood film
- parasitaemia > 2%
- hypoglycaemia
- acidosis
- temperature > 39 °C
- severe anaemia
- complications as below

Complications

- cerebral malaria: seizures, coma
- acute renal failure: blackwater fever, secondary to intravascular haemolysis, mechanism unknown
- acute respiratory distress syndrome (ARDS)
- hypoglycaemia
- disseminated intravascular coagulation (DIC)

Management

Uncomplicated falciparum malaria

- strains resistant to chloroquine are prevalent in certain areas of Asia and Africa
- the 2010 WHO guidelines recommend artemisinin-based combination therapies (ACTs) as first-line therapy
- examples include artemether plus lumefantrine, artesunate plus amodiaquine, artesunate plus mefloquine, artesunate plus sulfadoxine-pyrimethamine, dihydroartemisinin plus piperaquine

Severe falciparum malaria

- a parasite counts of more than 2% will usually need parenteral treatment irrespective of clinical state
- intravenous artesunate is now recommended by WHO in preference to intravenous quinine
- if parasite count > 10% then exchange transfusion should be considered
- shock may indicate coexistent bacterial septicaemia - malaria rarely causes haemodynamic collapse

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

WHO

9 7

Malaria treatment guidelines

Suggest link

Report broken link

Media



Malaria

Osmosis - YouTube

5 1

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A 30-year-old woman with rheumatoid arthritis has been diagnosed in the community with a prolonged urinary tract infection. For her rheumatoid arthritis she has been stable on azathioprine for nearly a year and besides paracetamol she takes no other regular medications.

The GP has requested a follow-up blood test which is shown below.

Baseline bloods were taken 2 months ago:

Hb	120 g/l
Platelets	310 * 10^9/l
WBC	4.5 * 10^9/l

Yesterday the bloods showed the following:

Hb	101 g/l
Platelets	296 * 10^9/l
WBC	1.9 * 10^9/l

Which of the following drugs is most likely responsible?

- Trimethoprim
- Nitrofurantoin
- Cephalexin
- Amoxicillin
- Co-amoxiclav

Submit answer

Reference ranges

Score: **16.7%**

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Which of the following drugs is most likely responsible?

Trimethoprim	77%
Nitrofurantoin	20%
Cephalexin	2%
Amoxicillin	1%
Co-amoxiclav	1%

It is likely she has received a protracted course of antibiotics, possibly more than one different antibiotic even.

An important side effect of azathioprine is cytopaenia. Azathioprine is metabolised to 6-mercaptopurine (6-MP) and then inactivated either by oxidation or methylation. Oxidation is catalysed by xanthine oxidase and methylation is catalysed by the enzyme TPMT. Factors that can lead to elevated levels of azathioprine are:

- Drugs that inhibit xanthine oxidase e.g. allopurinol

- Drugs that can also cause myelosuppression e.g.sulphonamides, trimethoprim
- Reduced activity of TPMT

Discuss (2) Improve

Next question

Trimethoprim

Trimethoprim is an antibiotic, mainly used in the management of urinary tract infections.

Mechanism of action

- interferes with DNA synthesis by inhibiting dihydrofolate reductase
  - may, therefore, interact with methotrexate, which also inhibits dihydrofolate reductase

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AI + Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

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A 70-year-old female was admitted to the emergency department from a nursing home due to a progressive decline in her level of consciousness over the past two days. Her carer mentioned that she had been complaining of a burning sensation while urinating associated with a low-grade fever for the last week. Blood investigations showed:

Na^+	130 mmol/l
K^+	3.6 mmol/l
Urea	13 mmol/l
Creatinine	130 µmol/l

Urine dipstick showed increase leukocytes and nitrites. Urine culture showed a growth of extended-spectrum B-lactamase (ESBL) - producing *Escherichia coli*.

What is the first line treatment?

- Ciprofloxacin
- Ceftriaxone
- Meropenem
- Methoprim
- Aztreonam

Submit answer

Reference ranges

Score: **16.7%**

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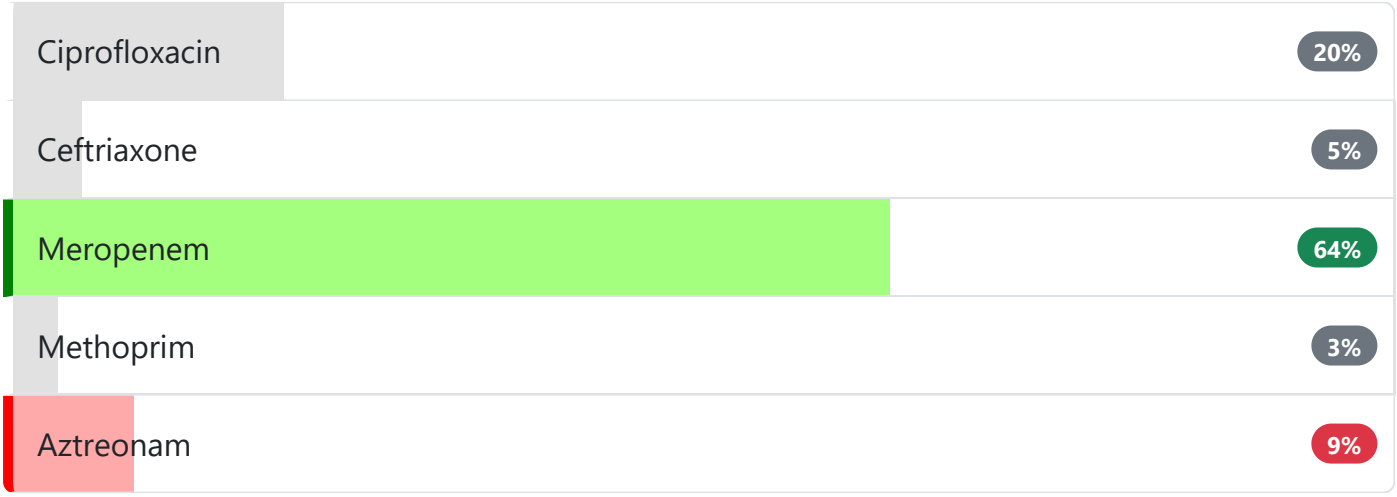
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A 70-year-old female was admitted to the emergency department from a nursing home due to a progressive decline in her level of consciousness over the past two days. Her carer mentioned that she had been complaining of a burning sensation while urinating associated with a low-grade fever for the last week. Blood investigations showed:

Na^+	130 mmol/l
K^+	3.6 mmol/l
Urea	13 mmol/l
Creatinine	130 µmol/l

Urine dipstick showed increase leukocytes and nitrites. Urine culture showed a growth of extended-spectrum B-lactamase (ESBL) - producing *Escherichia coli*.

What is the first line treatment?



The regular use of B-lactam antibiotics for urinary tract infection has resulted in the emergence of new strains of bacteria that are resistant to these antibiotics. ESBL is one of the most problematic strains that is usually difficult to treat.

First line antibiotics are still carbapenems (imipenem, meropenem, ertapenem, doripenem).

Giving these antibiotics may further increase resistance so the advice is not to give antibiotics unless there are clinical signs and symptoms of a urinary tract infection.

Source:
Shaikh, S., Fatima, J., Shakil, S., Rizvi, S. and Kamal, M. (2015). Antibiotic resistance and extended-spectrum beta-lactamases: Types, epidemiology and treatment. Saudi Journal of Biological Sciences, 22(1), pp.90-101.

Discuss (3) Improve

Next question

Escherichia coli

Escherichia coli is a facultative anaerobic, lactose-fermenting, Gram negative rod which is a normal gut commensal.

E. coli infections lead to a variety of diseases in humans including:

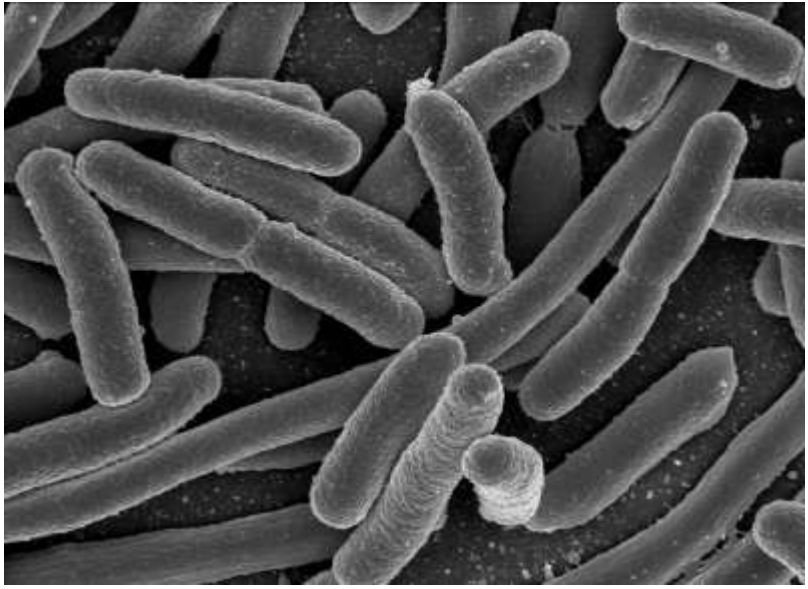
- diarrhoeal illnesses
- UTIs
- neonatal meningitis

Serotypes

E. coli may be classified according to the antigens which may trigger an immune response:

Antigen	Origin	Notes
O	Lipopolysaccharide layer	
K	Capsule	Neonatal meningitis secondary to *E. coli* is usually caused by a serotype that contains the capsular antigen K-1
H	Flagellin	

E. coli O157:H7 is a particular strain associated with severe, haemorrhagic, watery diarrhoea. It has a high mortality rate and can be complicated by haemolytic uraemic syndrome. It is often spread by contaminated ground beef.



Scanning electron micrograph of *Escherichia coli*, grown in culture and adhered to a cover slip. Credit: NIAID

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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Back to top

A 27-year-old nurse attends the emergency department and reports she has just suffered a significant needle-stick injury while caring for her patient on the intensive care unit. While providing her patient personal care with her colleague, she was injured in the hand by a wide bore stylet needle previously used to introduce an IV cannula to her patient that had not been placed in an appropriate sharps bin. The needle had been visibly blood stained. The nurse had been wearing gloves and had followed correct first aid procedure for needle-stick injuries.

The nurse was very concerned as the patient who had been the needle-stick donor was receiving treatment for the acute neurological phase of rabies. Review of the patients clinical notes (with the consent of the patients next of kin) indicated that he had contracted rabies following a dog bite two months previously in Pakistan. The diagnosis of rabies encephalitis had been made following clinical review by infectious disease experts and the detection of neutralising serum antibodies.

The nurse was previously fit and healthy with no significant past medical history. She did not use any regular medications and had no allergies. She was uncertain if she had had any previous vaccination against rabies, although did recall having a course of injections 10 years previously before she visited family in rural India.

What is the correct management of the needle-stick injury to prevent transmission of rabies?

- Rabies immunoglobulin and full course rabies vaccination
- Booster course rabies vaccination
- Rabies immunoglobulin
- Full course rabies vaccination
- No action required

Submit answer

Reference ranges

Score: **16.7%**

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The nurse was previously fit and healthy with no significant past medical history. She did not use any regular medications and had no allergies. She was uncertain if she had had any previous vaccination against rabies, although did recall having a course of injections 10 years previously before she visited family in rural India.

What is the correct management of the needle-stick injury to prevent transmission of rabies?

Rabies immunoglobulin and full course rabies vaccination	68%
Booster course rabies vaccination	6%
Rabies immunoglobulin	7%
Full course rabies vaccination	5%
No action required	15%

This situation represents a difficult clinical decision. Humans are considered to be an end-host for the rabies virus as inter-human transmission is not documented outside of transplantation cases. For that reason, only standard infection control precautions are recommended for health-care workers when in contact with individuals with rabies. However, the needle-stick injury described could foreseeably lead to transmission of the virus and given the severe consequences of rabies should be considered as possible rabies exposure.

The patient does not have a clear history of previous immunisation (the history stated could also be consistent with hepatitis B vaccination for example). Rabies immunoglobulin should be given to provide passive antibodies at the site of exposure prior to the development of an immune

response. As far as possible, the dose (20 IU / Kg) should be administered locally around the wound. In addition, post-exposure vaccination should be provided, with the WHO currently recommending a four dose schedule (given on days 0, 3, 7 and 14).

If an individual has been previously vaccinated then immunoglobulin is not required and a two dose vaccination schedule (given on days 0 and 3) should be given.

Crowcroft N, Thampi N. The prevention and management of rabies. BMJ 2015;350:g7827.

Discuss (4) Improve

Next question >

Rabies

Rabies is a viral disease that causes an acute encephalitis. The rabies virus is classed as a RNA rhabdovirus (specifically a lyssavirus) and has a bullet-shaped capsid. The vast majority of cases are caused by dog bites but it may also be transmitted by bat, raccoon and skunk bites.

After entry, the virus replicates in muscle tissue near the site of the bite before entering the peripheral nervous system. It then travels up to the central nervous system, where it causes encephalitis, leading to severe neurological symptoms. The virus eventually reaches the salivary glands, facilitating transmission.

Rabies is estimated to still kill around 25,000-50,000 people across the world each year. The vast majority of the disease burden falls on people in poor rural areas of Africa and Asia. Children are particularly at risk.

Features

- prodrome: headache, fever, agitation
- hydrophobia: water-provoking muscle spasms
- hypersalivation
- Negri bodies: cytoplasmic inclusion bodies found in infected neurons

There is now considered to be 'no risk' of developing rabies following an animal bite in the UK and the majority of developed countries. Following an animal bite in at-risk countries:

- the wound should be washed
- if an individual is already immunised then 2 further doses of vaccine should be given
- if not previously immunised then human rabies immunoglobulin (HRIG) should be given along with a full course of vaccination. If possible, the dose should be administered locally around the wound

If untreated the disease is nearly always fatal.

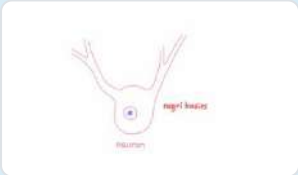
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Rabies

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Score: **16.7%**

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Back to top

A 65-year-old man is referred to gastroenterology clinic for advice prior to planned overseas travel. The patient states that he is planning to visit relatives in Brazil in two months time and is very concerned about the possibility of contracting travellers' diarrhoea in light of his previous and current medical history.

The patient had undergone a total colectomy with ileostomy formation twenty years previously, as curative treatment for severe ulcerative colitis. He had subsequently learnt to manage his ileostomy well and had declined reconstructive surgery. Ten years previously, the patient had contracted diarrhoea while travelling in the Caribbean, which he reported had ruined his holiday and complicated his ileostomy management for weeks.

Of more pressing concern was a recent diagnosis of small-cell lung cancer, for which the patient was undergoing chemotherapy with cisplatin and etoposide. The patient's chemotherapy regime was due to be completed four weeks before his proposed travel.

Aside from the issues outlined above, the patient's only other past medical history included hypertension for which he took ramipril. The patient denied any drug allergies or intolerances.

Standard precautions regarding the avoidance of travellers' diarrhoea were discussed with the patient, who fully acknowledged the risks of travelling so soon after completing chemotherapy.

What is appropriate management to help prevent travellers' diarrhoea in this patient?

- *Vibrio cholerae* vaccination
- Standard advice regarding hygiene precautions only
- Co-amoxiclav prophylaxis while overseas
- Ciprofloxacin prophylaxis while overseas
- Low-dose loperamide

Submit answer

Reference ranges

Score: **16.7%**

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What is appropriate management to help prevent travellers' diarrhoea in this patient?

Vibrio cholerae vaccination	9%
Standard advice regarding hygiene precautions only	56%
Co-amoxiclav prophylaxis while overseas	2%
Ciprofloxacin prophylaxis while overseas	30%
Low-dose loperamide	3%

Travellers' diarrhoea is most commonly caused by *Escherichia coli* species and *Campylobacter jejuni*. Less common causative pathogens include *Salmonella* and *Shigella* species, parasitic infections such as *Giardia lamblia*, and viruses such as norovirus.

In the majority of individuals, diarrhoea is annoying and unpleasant but does not lead to long-term sequelae. However, some groups of individuals may be unable to tolerate the consequences of dehydration from diarrhoea, or be vulnerable to invasive complications such as bacteraemia.

The patient presented in this question will be immunosuppressed secondary to his chemotherapy and has an under-lying bowel condition due to his ileostomy. Therefore, he should be considered for antibiotic prophylaxis to prevent travellers' diarrhoea, following discussion of the risks of antibiotic associated diarrhoea and other side effects.When antibiotic prophylaxis is used, typical choices of agent are ciprofloxacin, norfloxacin or rifaximin.

Anti-motility agents such as loperamide can be helpful in limiting diarrhoea in individuals unable to tolerate dehydration, but does not have a role in preventing infection. Symptoms of invasive colitis, such as severe abdominal pain or bloody diarrhoea, are contra-indications to the use of anti-motility agents due to the risk of intestinal perforation. Vaccination against cholera is effective, however is not a common cause of travellers' diarrhoea.

Barrett J, Brown M. Travellers' diarrhoea. BMJ 2016;353:i1937.

Discuss (7) | Improve

Next question >

Gastroenteritis: causes

Gastroenteritis may either occur whilst at home or whilst travelling abroad (travellers' diarrhoea)

Travellers' diarrhoea may be defined as at least 3 loose to watery stools in 24 hours with or without one of more of abdominal cramps, fever, nausea, vomiting or blood in the stool. The most common cause is *Escherichia coli*.

Another pattern of illness is 'acute food poisoning'. This describes the sudden onset of nausea, vomiting and diarrhoea after the ingestion of a toxin. Acute food poisoning is typically caused by *Staphylococcus aureus*, *Bacillus cereus* or *Clostridium perfringens*.

Stereotypical histories

Infection	Typical presentation
Escherichia coli	Common amongst travellers Watery stools Abdominal cramps and nausea
Giardiasis	Prolonged, non-bloody diarrhoea

Infection	Typical presentation
Cholera	Profuse, watery diarrhoea Severe dehydration resulting in weight loss Not common amongst travellers
Shigella	Bloody diarrhoea Vomiting and abdominal pain
Staphylococcus aureus	Severe vomiting Short incubation period
Campylobacter	A flu-like prodrome is usually followed by crampy abdominal pains, fever and diarrhoea which may be bloody May mimic appendicitis Complications include Guillain-Barre syndrome
Bacillus cereus	Two types of illness are seen • vomiting within 6 hours, stereotypically due to rice • diarrhoeal illness occurring after 6 hours
Amoebiasis	Gradual onset bloody diarrhoea, abdominal pain and tenderness which may last for several weeks

Incubation period

- 1-6 hrs: *Staphylococcus aureus*, *Bacillus cereus**
- 12-48 hrs: *Salmonella*, *Escherichia coli*
- 48-72 hrs: *Shigella*, *Campylobacter*
- > 7 days: Giardiasis, Amoebiasis

*vomiting subtype, the diarrhoeal illness has an incubation period of 6-14 hours

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 16 13

2011 Diarrhoeal disease review

Suggest link Report broken link

Media



Shigella

ATP - YouTube 5 0



Salmonella

ATP - YouTube 5 0

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A 19-year-old man attends the Emergency department with a fever for the past one week. He notes that the fever comes on alternate days. He reports also a co-existing headache, but no other particular symptoms. Of note, he returned from Afghanistan two weeks ago. Examination reveals a temperature of 38.1ºC, heart rate 89/min, blood pressure 123/78 mmHg, respiratory rate 17/min and oxygen saturations of 99%. There is no evidence of no neck stiffness, objective photophobia, jaundice or splenomegaly.

What is the most likely diagnosis?

- Chikungunya
- Zika virus
- Plasmodium vivax
- Plasmodium falciparum
- Dengue fever

Submit answer

Reference ranges

Score: **16.7%**

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What is the most likely diagnosis?

Chikungunya	6%
Zika virus	7%
Plasmodium vivax	57%
Plasmodium falciparum	20%
Dengue fever	10%

Fever in the returning traveller is a common presentation in the Emergency department. A knowledge of the areas travelled and incubation periods is essential to accurately diagnosing the potential infectious disease.

Chikungunya is a viral disease transmitted to humans via infected mosquitos. Fever and joint pain is the mos common presentation. The incubation period is 2-12 days.

Zika virus is currently not present in Afghanistan.

Dengue fever has a incubation period of 4-10 days.

This leaves plasmodium falciparum, which has a usual incubation of 7-14 days (but up to 3 months), and plasmodium vivax which has a typical incubation of 12-17 days (but reported up to 12 months) following exposure. Factors that point away from p. falciparum are the milder presentation and the much lower prevalence of it in Afghanistan (p. falciparum 5%, p. vivax 95%). Furthermore, fever due to malaria on alternate days, known as benign tertian malaria, is suggestive of either p. vivax or p. ovale. Comparatively, p. falciparum will typically cause more of a continuous fever.

Next question

Malaria: non-falciparum

The most common cause of non-falciparum malaria is *Plasmodium vivax*, with *Plasmodium ovale* and *Plasmodium malariae* accounting for the other cases. *Plasmodium vivax* is often found in Central America and the Indian Subcontinent whilst *Plasmodium ovale* typically comes from Africa.

Plasmodium knowlesi is another non-falciparum species which causes clinical pathology, found predominantly in South East Asia.

Features

- general features of malaria: fever, headache, splenomegaly
- *Plasmodium vivax/ovale*: cyclical fever every 48 hours. *Plasmodium malariae*: cyclical fever every 72 hours
- *Plasmodium malariae*: is associated with nephrotic syndrome.

Ovale and vivax malaria have a hypnozoite stage and may therefore relapse following treatment.

Treatment

- in areas which are known to be chloroquine-sensitive then WHO recommend either an artemisinin-based combination therapy (ACT) or chloroquine
- in areas which are known to be chloroquine-resistant an ACT should be used
- ACTs should be avoided in pregnant women
- patients with ovale or vivax malaria should be given primaquine following acute treatment with chloroquine to destroy liver hypnozoites and prevent relapse

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

WHO

9 1

Malaria treatment guidelines

Suggest link

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Media



Malaria

Osmosis - YouTube

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Score: **16.7%**

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A 76-year-old female has been under the care of the medical team for the past week. She has been treated for a full sensitive *E. coli* bacteraemia with intravenous co-amoxiclav thought be sourced from the urinary system. On completing the course, the patient remains in for ongoing physiotherapy input. The next day, the patient has a temperature spike and cultures demonstrate regrowth of *E. coli*.

What is the most appropriate next step?

- Escalate antibiotics to meropenem
- Image the renal tract
- Commence treatment for hospital-acquired pneumonia
- Urology referral
- Urgent nephrostomy

Submit answer

Reference ranges

Score: **16.7%**

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A 76-year-old female has been under the care of the medical team for the past week. She has been treated for a full sensitive *E. coli* bacteraemia with intravenous co-amoxiclav thought be sourced from the urinary system. On completing the course, the patient remains in for ongoing physiotherapy input. The next day, the patient has a temperature spike and cultures demonstrate regrowth of *E. coli*.

What is the most appropriate next step?

Escalate antibiotics to meropenem	37%
Image the renal tract	53%
Commence treatment for hospital-acquired pneumonia	4%
Urology referral	6%
Urgent nephrostomy	0%

Discussion of this case with microbiology would likely result in the suggestion of imaging. In someone who has appropriate antimicrobial treatment, regrowth of the same organism would not be expected so soon. This may indicate there is a source of the bacteria that is not being adequately treated - a renal abscess in this case, which may be identified on ultrasound.

Escalation to meropenem should not be considered necessary if the organism has been fully sensitive to the narrower spectrum antibiotics.

E. coli would be an unlikely source of hospital-acquired pneumonia.

Urology referral would be the next step should a renal abscess be identified.

Imaging should first be carried out to determine whether a nephrostomy is required.

Discuss (5) | Improve

Next question >

Escherichia coli

Escherichia coli is a facultative anaerobic, lactose-fermenting, Gram negative rod which is a normal gut commensal.

E. coli infections lead to a variety of diseases in humans including:

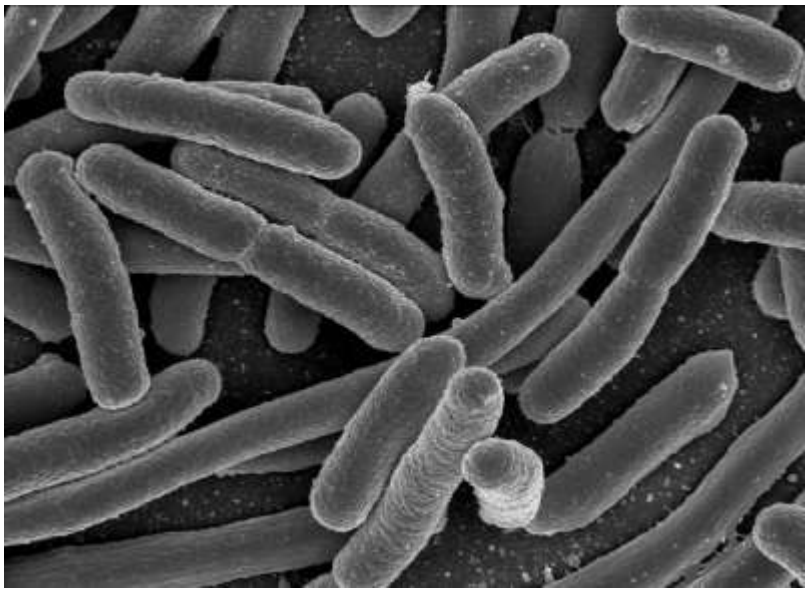
- diarrhoeal illnesses
- UTIs
- neonatal meningitis

Serotypes

E. coli may be classified according to the antigens which may trigger an immune response:

Antigen	Origin	Notes
O	Lipopolysaccharide layer	
K	Capsule	Neonatal meningitis secondary to *E. coli* is usually caused by a serotype that contains the capsular antigen K-1
H	Flagellin	

E. coli O157:H7 is a particular strain associated with severe, haemorrhagic, watery diarrhoea. It has a high mortality rate and can be complicated by haemolytic uraemic syndrome. It is often spread by contaminated ground beef.



Scanning electron micrograph of *Escherichia coli*, grown in culture and adhered to a cover slip. Credit: NIAID

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 26-year-old man presents with an intensely itchy rash which he thinks is spreading rapidly. He has recently returned from a holiday in Brazil, and mentions having seen many stray cats around the hotel resort. Symptoms began approximately 1 week after returning from the holiday. He denies having had any similar rashes previously, has no significant past medical history and is systemically well.

On examination, the rash appears as follows:



Given the most likely diagnosis, which of the following is the most appropriate management?

- Doxycycline
- Hydrocortisone
- Ivermectin
- Malathion
- Permethrin

Submit answer

Reference ranges

Score: **16.7%**

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On examination, the rash appears as follows:



Given the most likely diagnosis, which of the following is the most appropriate management?

Doxycycline	16%
Hydrocortisone	1%
Ivermectin	74%
Malathion	3%
Permethrin	6%

The rash above has the appearance of cutaneous larva migrans, most commonly caused by infection with the cat hookworm *Ancyclostoma braziliense*. It is found in tropical regions throughout the world, including Brazil, from where this patient has recently returned, having

mentioned a resort with many stray cats. The rash creeps due to the migration of the larvae. The photo above demonstrates this creeping, serpiginous eruption. Management involves the use of ivermectin, which is the correct answer here, or albendazole.

Doxycycline is incorrect. This is an antibiotic that would be used for the management of Lyme disease. This would instead present with erythema chronicum migrans - annular, bulls-eye lesions, rather than the tortuous lesions seen here.

Hydrocortisone is a topical steroid and is not involved in the management of cutaneous larva migrans. Ivermectin or albendazole should be given.

Malathion is a second-line option for the management of scabies. Whilst also presenting with intense pruritus, scabies lesions would instead appear as small, discrete burrows, particularly in the web spaces between fingers, as well as the palms and wrists. The serpiginous lesions seen here are more indicative of cutaneous larva migrans.

Permethrin is generally the first-line treatment used for scabies. Again, the lesions seen above are more associated with cutaneous larva migrans, not scabies.

Discuss (4) | Improve

Next question >

Cutaneous larva migrans

Cutaneous larva migrans is a dermatological condition prevalent in tropical and subtropical regions, largely attributable to cutaneous penetration and subsequent migration of nematode larvae, primarily from the *Ancylostoma* genus (e.g. *Ancyclostoma braziliense*). Typically, the transmission vectors are faecal-contaminated soil or sand, posing significant risks to individuals with a history of barefoot beach visits or direct soil contact.

The clinical presentation typically involves an intensely pruritic, 'creeping', serpiginous, erythematous cutaneous eruption that advances over time. Symptoms can last for weeks to months, potentially leading to secondary bacterial infection due to excessive scratching. Diagnosis is typically clinical, based on exposure history and characteristic skin manifestations.



Treatment options revolve around anthelmintic agents, such as ivermectin or albendazole. Topical

therapy with thiabendazole can also be effective, although it's generally less preferred due to a lower efficacy rate and higher side effect profile.

Prevention strategies largely involve patient education about appropriate protective measures, particularly avoiding direct skin contact with potentially contaminated soil. Given the zoonotic nature of cutaneous larva migrans, public health measures for animal defecation control can also contribute to reducing the prevalence of this condition.



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Center for Disease Control

3 2

Cutaneous Larva Migrans

Suggest link

Report broken link

Score: **16.7%**

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A 44-year-old businessman was admitted to the general medical take with fever, jaundice and headaches. He had returned from a business trip to Hong Kong one month ago. He is a keen river sailor and has been unable to sail for the last two weeks.

On examination, he is tachycardic with a temperature of 39.5°C. He appears confused and is combative during the examination. His chest has scattered crackles bibasally with some right upper quadrant abdominal pain

Investigations

Chest X-Ray	Hazy lung infiltrates bibasally

Haemoglobin	135 g/L (130-180)
White cell count	17.8 Ã— 109/L (4.0-11.0)
Neutrophil count	15.7 Ã— 109/L (1.5-7.0)
Platelet count	413 Ã— 109/L (150-400)

Serum C-reactive protein	345 mg/L (<10)
Serum urea	13.3 mmol/L (2.5-7.0)
Serum total bilirubin	110 mol/L (1-22)
Serum alanine aminotransferase	117 U/L (5-35)
Serum alkaline phosphatase	110 U/L (45-105)
Serum gamma glutamyl transferase	61 U/L (<50)

What is the most likely diagnosis?

- Dengue Fever
- HIV seroconversion
- Acute hepatitis A
- Malaria
- Leptospirosis

Submit answer

Reference ranges

Score: **16.7%**

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What is the most likely diagnosis?

Dengue Fever	7%
HIV seroconversion	8%
Acute hepatitis A	9%
Malaria	4%
Leptospirosis	72%

Leptospirosis is the most likely diagnosis. The river sailing puts this gentleman at risk of contact with contaminated water from animal urine. Incubation is between 4-14 days. Typical symptoms include high fever, headache, muscle aches, vomiting and may have meningitis and jaundice.

Hong Kong is not a malaria endemic region nor is it a common region for Dengue Fever. HIV seroconversion would not explain jaundice or deranged liver function. Acute hepatitis A infection is prevalent in Hong Kong and incubation period is between 2 - 6 weeks but infection would not explain the neurological symptoms.

Discuss (9) | Improve

Next question >

Leptospirosis ★

Leptospirosis is caused by the spirochaete *Leptospira interrogans* (serogroup *L. icterohaemorrhagiae*), classically being spread by contact with infected rat urine.

Epidemiology

- leptospirosis is commonly seen in questions referring to sewage workers, farmers, vets or people who work in an abattoir
- however, on an international level, leptospirosis is far more common in the tropics so should be considered in the returning traveller

Weil's disease should always be considered in high-risk patients with hepatorenal failure

Features

- the early phase is due to bacteraemia and lasts around a week
  - may be mild or subclinical
  - fever
  - flu-like symptoms
  - subconjunctival suffusion (redness)/haemorrhage
- second immune phase may lead to more severe disease (Weil's disease)
  - acute kidney injury (seen in 50% of patients)
  - hepatitis: jaundice, hepatomegaly
  - aseptic meningitis

Investigation

- **serology**: antibodies to *Leptospira* develop after about 7 days
- PCR

- culture
  - growth may take several weeks so limits usefulness in diagnosis
  - blood and CSF samples are generally positive for the first 10 days
  - urine cultures become positive during the second week of illness

Management
- high-dose benzylpenicillin or doxycycline

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Quarterly Journal of Medicine 12 4

Leptospirosis review

Suggest link Report broken link

Media



Leptospira

Osmosis - YouTube 7 0

Report broken media

Score: **16.7%**

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A 23 year-old artist presents after he woke up with headache, neck stiffness, and photophobia. He is normally fit and well, but for the last three weeks has complained of feeling tired and irritable.

On examination, the left side of the palate does not elevate, and the tongue is deviated to the left upon protrusion. The remainder of the neurological examination is unremarkable.

Plain computed tomography of the head is unremarkable.

Lumbar puncture is performed with results of CSF analysis as follows:

Appearance	Turbid
White blood cells	28 cells/mm³ (95% lymphocytes)
Red blood cells	<1 cells/mm³
Gram stain	No organisms seen
Protein	1.32 g/L
Glucose	1.4 mmol/L
Serum glucose	7.5 mmol/L

What is the most likely diagnosis?

- Meningococcal meningitis
- Tuberculous meningitis
- Subarachnoid haemorrhage
- Skull base tumour
- Malignant meningitis

Submit answer

Reference ranges

Score: **16.7%**

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What is the most likely diagnosis?

Meningococcal meningitis	10%
Tuberculous meningitis	75%
Subarachnoid haemorrhage	1%
Skull base tumour	3%
Malignant meningitis	10%

The prodromal phase of malaise and possible personality change, followed by meningism, with the onset of basal cranial nerve palsies, and coupled with the CSF findings of lymphocytic pleocytosis, raised protein, and low glucose, are all highly suggestive of tuberculous meningitis.

There is no particular suggestion of a primary malignancy, but malignant meningitis (leptomeningeal carcinomatosis) is a possibility and it would be reasonable to send a large volume of CSF for cytology. Like tuberculous meningitis, this condition typically presents with headache, meningism, and cranial nerve palsies. However, it is eye movements which are most often affected.

There is often also invasion of spinal meninges which involves nerve roots and mimics radiculopathy. The CSF is hardly ever normal, although there are no specific features other than the direct demonstration of malignant cells.

Meningococcal meningitis is an acute disease, so the three week prodrome described here would be unusual. The presence of cranial nerve palsies would be extremely unusual. The CSF would be expected to show a neutrophilic pleocytosis, with a low glucose (less that 50% of serum glucose), but not as low as in tuberculous meningitis.

Subarachnoid haemorrhage should be considered in any case of acute headache. The normal CT scan is reassuring but does not exclude the diagnosis, but in any case the CSF is not suggestive.

A tumour of the skull base might explain the lower cranial nerve palsies, but one would have expected some more suggestive symptoms and an abnormal CT head. This diagnosis would not explain the grossly abnormal CSF findings.

Discuss (4) | Improve

Next question >

Meningitis: CSF analysis ★

The table below summarises the characteristic cerebrospinal fluid (CSF) findings in meningitis:

	Bacterial	**Viral**	**Tuberculous**	**Fungal**
Appearance	Cloudy	Clear/cloudy	Slight cloudy, fibrin web	Cloudy
Glucose	Low (< 1/2 plasma)	60-80% of plasma glucose*	Low (< 1/2 plasma)	Low
Protein	High (> 1 g/l)	Normal/raised	High (> 1 g/l)	High
White cells	10 - 5,000 polymorphs/mm^3	15 - 1,000 lymphocytes/mm^3	30 - 300 lymphocytes/mm^3	20 - 200 lymphocytes/mm^3

The Ziehl-Neelsen stain is only 20% sensitive in the detection of tuberculous meningitis and therefore PCR is sometimes used (sensitivity = 75%)

*mumps is unusual in being associated with a low glucose level in a proportion of cases. A low glucose may also be seen in herpes encephalitis

Next question

Textbooks

High-yield textbook

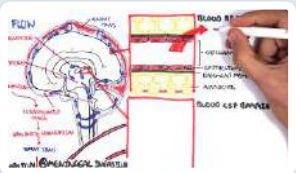
Extended textbook

Media



Meningitis

Osmosis - YouTube 3 0



(Bacterial) Meningitis Pathophysiology

Armando Hasudungan - YouTube 2 3

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A 38-year-old man is referred in by his GP with increasing frequency of diarrhoea over the last 4 days. He is a known ulcerative colitis patient who is well managed on azathioprine 200mg OD and mesalazine 2.4mg BD. He last had a flare 2 years ago and has been well in the interim. He is passing up to 10 watery motions a day and is suffering from faecal urgency and nocturnal episodes. He describes cramping left iliac fossa pain. There is no blood or mucus in the stools. On examination, he is febrile at 38.2 degrees celsius. His blood pressure is 120/75 mmHg and his heart rate is 100/min. On examination, he is underweight with a BMI of 18.5 and dehydrated. His abdomen is soft but he is tender in the left iliac fossa. He refuses a PR examination. Respiratory and cardiovascular examinations are normal.

His blood tests show:

Hb	110 g/l
Platelets	189 * 10^9/l
WBC	3.8 * 10^9/l
Neutrophils	0.89 * 10^9/l
INR	1.1 (0.9-1.2)

Na^+	136 mmol/l
K^+	4.9 mmol/l
Urea	8.0 mmol/l
Creatinine	100 µmol/l
Mag nesium	0.79 mmol/L (0.7-1.0)
Calcium	2.4 mmol/L (2.1-2.58)
CRP	78 mg/l

Bilirubin	5 µmol/l
ALP	78 u/l
ALT	28 u/l
Albumin	33 g/l

He is started on IV hydrocortisone 100mg QDS and IV fluids. Stool specimens are sent and are reported as negative for *C. difficile* toxin. He has a flexible sigmoidoscopy the next day which shows widespread left sided colitis. The biopsy results show the presence of inclusion bodies in the colonic mucosa. What is the appropriate treatment to start for this gentleman?

- Infliximab
- Ganciclovir
- Metronidazole
- Fluconazole
- Tazocin

Submit answer

Reference ranges

Score: **16.7%**

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A 38-year-old man is referred in by his GP with increasing frequency of diarrhoea over the last 4 days. He is a known ulcerative colitis patient who is well managed on azathioprine 200mg OD and mesalazine 2.4mg BD. He last had a flare 2 years ago and has been well in the interim. He is passing up to 10 watery motions a day and is suffering from faecal urgency and nocturnal episodes. He describes cramping left iliac fossa pain. There is no blood or mucus in the stools. On examination, he is febrile at 38.2 degrees celsius. His blood pressure is 120/75 mmHg and his heart rate is 100/min. On examination, he is underweight with a BMI of 18.5 and dehydrated. His abdomen is soft but he is tender in the left iliac fossa. He refuses a PR examination. Respiratory and cardiovascular examinations are normal.

His blood tests show:

Hb	110 g/l
Platelets	189 * 10^9/l
WBC	3.8 * 10^9/l
Neutrophils	0.89 * 10^9/l
INR	1.1 (0.9-1.2)

Na^+	136 mmol/l
K^+	4.9 mmol/l
Urea	8.0 mmol/l
Creatinine	100 µmol/l
Mag nesium	0.79 mmol/L (0.7-1.0)
Calcium	2.4 mmol/L (2.1-2.58)
CRP	78 mg/l

Bilirubin	5 µmol/l
ALP	78 u/l
ALT	28 u/l
Albumin	33 g/l

He is started on IV hydrocortisone 100mg QDS and IV fluids. Stool specimens are sent and are reported as negative for *C. difficile* toxin. He has a flexible sigmoidoscopy the next day which shows widespread left sided colitis. The biopsy results show the presence of inclusion bodies in the colonic mucosa. What is the appropriate treatment to start for this gentleman?

Infliximab	13%
Ganciclovir	60%
Metronidazole	21%
Fluconazole	4%
Tazocin	3%

This patient has CMV colitis. It can be a consequence of immunosuppressive agents such as azathioprine. It can present with fever and diarrhoea +/- blood. It can also present as an exacerbation of IBD patients in those on immunosuppressants. Inclusion bodies are characteristic at biopsy. It will respond well to an antiviral agent and ganciclovir is the treatment necessary here. Tazocin, metronidazole and fluconazole will not treat appropriately and infliximab would only be indicated if a colitis flare was not due to an infective cause.

Discuss (5) Improve

Next question

Cytomegalovirus

Cytomegalovirus (CMV) is one of the herpes viruses. It is thought that around 50% of people have been exposed to the CMV virus although it only usually causes disease in the immunocompromised, for example people with HIV or those on immunosuppressants following organ transplantation.

Pathophysiology

- infected cells have a 'Owl's eye' appearance due to intranuclear inclusion bodies

Patterns of disease

Congenital CMV infection

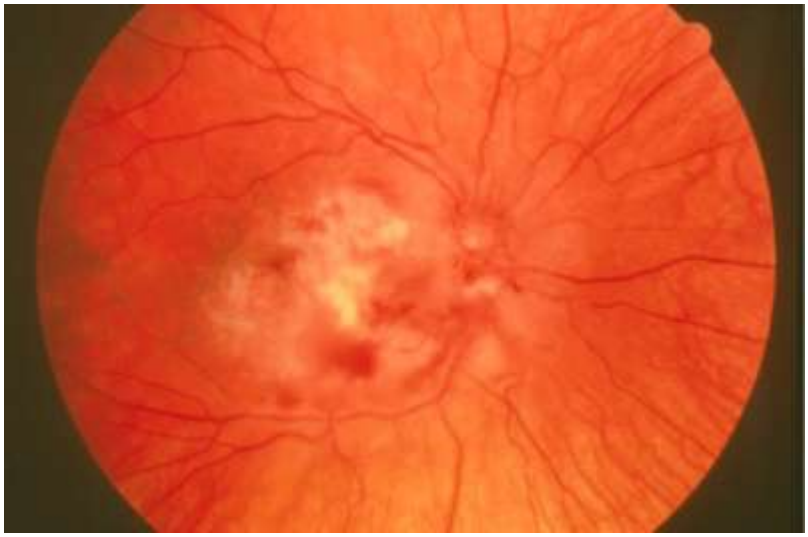
- features include growth retardation, pinpoint petechial 'blueberry muffin' skin lesions, microcephaly, sensorineural deafness, encephalitiis (seizures) and hepatosplenomegaly

CMV mononucleosis

- infectious mononucelosis-like illness
- may develop in immunocompetent individuals

CMV retinitis

- common in HIV patients with a low CD4 count (< 50)
- presents with visual impairment e.g. 'blurred vision'. Fundoscopy shows retinal haemorrhages and necrosis, often called 'pizza' retina
- IV ganciclovir is the treatment of choice



Fundus photograph showing CMV retinitis. Credit: National Eye Institute, National Institutes of Health

CMV encephalopathy

- seen in patients with HIV who have low CD4 counts

CMV pneumonitis

CMV colitis

AI 123

Next question ›

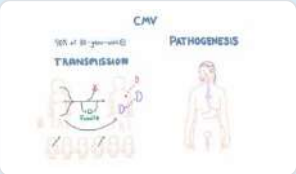
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Textbooks

High-yield textbook

Extended textbook

Media



CMV - Mono, Congenital Infection, Immunocompromised Hosts

macrophage - YouTube 4 0



CMV infection after transplant

Osmosis - YouTube 4 2

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A 47-year-old man is referred by the emergency department with increasing shortness of breath. He is a known HIV patient managed by the local GUM consultants. He is not on any antiretroviral treatment. He presents with a two-week history of worsening shortness of breath. He is now breathless on minimal exercise. He has a non-productive cough and has felt lethargic for the last week. On examination he is afebrile, his blood pressure is 120/89 mmHg and he is slightly tachycardic at 110bpm. His respiratory rate is 18 at rest with saturations on 98% on air. When he mobilises to the toilet he becoming very tachypnoeic and his saturations drop to 90%. His blood tests are as follows:

Hb	110 g/l
Platelets	201 * 10^9/l
WBC	9.6 * 10^9/l
Neutrophils	4.5 * 10^9/l

Na^+	138 mmol/l
K^+	4.1 mmol/l
Urea	7.8 mmol/l
Creatinine	20 µmol/l
CRP	70 mg/l

Bilirubin	5 µmol/l
ALP	89 u/l
ALT	43 u/l
Albumin	34 g/l

An ABG is done which shows

pH	7.35
pO2	7.7
pCO2	4.6
HCO3-	21
BE	-3
Lactate	2.2

His chest x-ray shows fine bilateral reticular nodular shadowing.

What is the most appropriate treatment to start in this gentleman?

- ○ Tazocin ×
- ○ Co-trimoxazole ×
- ○ Aciclovir ×
- ○ Co-trimoxazole and corticosteroids ×
- ○ Highly active anti-retroviral treatment ×

Submit answer

Reference ranges

Score: **16.7%**

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pCO2	4.6
HCO3-	21
BE	-3
Lactate	2.2

His chest x-ray shows fine bilateral reticular nodular shadowing.

What is the most appropriate treatment to start in this gentleman?

Tazocin	1%
Co-trimoxazole	24%
Aciclovir	1%
Co-trimoxazole and corticosteroids	69%
Highly active anti-retroviral treatment	5%

This gentleman is immunocompromised and has PCP. Patients with moderate to severe PCP should be treated with Co-trimoxazole and corticosteroids. Patients with mild to moderate PCP need only co-trimoxazole.

Mild:

- Breathlessness on mild exercise, which may be associated with a cough and sweats
- Arterial blood gases and oxygen saturation at rest, on air: PaO2 >11 kPa; SaO2 >96%
- Chest X-ray: normal or minor perihilar infiltrates

Moderate:

- Breathlessness on minimal exercise, fever (with or without sweats)
- Arterial blood gases and oxygen saturation at rest, on air: PaO2 8-11 kPa; SaO2 91-96%
- Chest X-ray: diffuse interstitial shadowing

Severe:

- Breathlessness at rest, persistent fever and cough
- Arterial blood gases and oxygen saturation at rest, on air: PaO2 <8 kPa; SaO2 <91%
- Chest X-Ray: extensive interstitial shadowing, with or without alveolar shadowing

👍 👎 💬 Discuss (6) Improve

Next question ›

HIV: *Pneumocystis jiroveci* pneumonia ★

Whilst the organism *Pneumocystis carinii* is now referred to as *Pneumocystis jiroveci*, the term *Pneumocystis carinii* pneumonia (PCP) is still in common use

- *Pneumocystis jiroveci* is an unicellular eukaryote, generally classified as a fungus but some authorities consider it a protozoa
- PCP is the most common opportunistic infection in AIDS
- all patients with a CD4 count < 200/mm^3 should receive PCP prophylaxis

Features

- dyspnoea
- dry cough
- fever
- very few chest signs

Pneumothorax is a common complication of PCP.

Extrapulmonary manifestations are rare (1-2% of cases), may cause

- hepatosplenomegaly
- lymphadenopathy
- choroid lesions

Investigation

- CXR: typically shows bilateral interstitial pulmonary infiltrates but can present with other x-ray findings e.g. lobar consolidation. May be normal
- exercise-induced desaturation
- sputum often fails to show PCP, bronchoalveolar lavage (BAL) often needed to demonstrate PCP (silver stain shows characteristic cysts)





Management

- co-trimoxazole
- IV pentamidine in severe cases
- aerosolized pentamidine is an alternative treatment for *Pneumocystis jiroveci* pneumonia but is less effective with a risk of pneumothorax
- steroids if hypoxic (if pO2 < 9.3kPa then steroids reduce risk of respiratory failure by 50% and death by a third)

AI + Q 123

Next question

Textbooks

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Extended textbook

Links

BNF

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Pneumocystis pneumonia

Patient.info

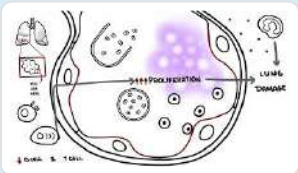
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Pneumocystis Jirovecii Pneumonia review

Suggest link

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Media



Pneumocystis jirovecii - Pneumocystis pneumonia

Armando Hasudungan - YouTube

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Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

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A 38-year-old man is admitted to the Emergency Department with shortness-of-breath and a non-productive cough. He has recently emigrated from Russia and has just started anti-retroviral therapy for HIV.

Examination of his chest is unremarkable. His temperature is 37.7ºC and oxygen saturations are 95% on room air.

A chest x-ray is performed:



© Image used on license from Radiopaedia

What is the most likely diagnosis?

- Cytomegalovirus
- Mediastinal lymphoma
- Tuberculosis
- Mycoplasma pneumonia
- Pneumocystis jiroveci pneumonia

Submit answer

Reference ranges

Score: **16.7%**

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What is the most likely diagnosis?

Cytomegalovirus	5%
Mediastinal lymphoma	1%
Tuberculosis	10%
Mycoplasma pneumonia	4%
Pneumocystis jiroveci pneumonia	80%

The key to answering this question is to look at the history rather than the x-ray. A combination of HIV + chest symptoms + unremarkable auscultatory findings in an exam are highly suggestive of Pneumocystis jiroveci pneumonia (more commonly known as PCP). X-ray changes in Pneumocystis jiroveci pneumonia are generally too subtle for non-radiologists to detect. The x-ray should however help you to exclude some of the other diagnoses.

The chest x-ray shows hazy, perihilar mid and upper zone opacification with some interstitial prominence. A few discrete cysts (termed pneumatocoeles) measuring up to 1cm are present. The presence of pneumatocoeles in a HIV patient suggests Pneumocystis jiroveci pneumonia.

👍 👎 💬 Discuss (5) Improve

Next question ›

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AI + Q 123

Next question >

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Textbooks

High-yield textbook

Extended textbook

Links

BNF 7 8

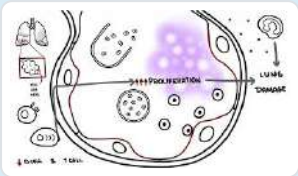
Pneumocystis pneumonia

Patient.info 6 6

Pneumocystis Jirovecii Pneumonia review

Suggest link Report broken link

Media



Pneumocystis jirovecii - Pneumocystis pneumonia

Armando Hasudungan - YouTube 0 0



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube 2 1

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A 53-year-old lady comes into the Emergency Department with a cough productive of green sputum and palpitations. She feels very unwell, feverish and lethargic. On examination, she has bronchial breathing at her right base with respiratory rate 25/min, sats 95% on room air. Her heart sounds are normal with an irregularly irregular heartbeat. Her heart rate was 120/min and blood pressure 90/40 mmHg. An ECG shows atrial fibrillation with a fast ventricular rate. She has no history of atrial fibrillation. What is the first treatment that should be given for her atrial fibrillation?

- Bisoprolol
- Digoxin
- Intravenous fluids
- Oral antibiotics
- Flecainide

Submit answer

Reference ranges

Score: **16.7%**

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Bisoprolol	11%
Digoxin	6%
Intravenous fluids	58%
Oral antibiotics	15%
Flecainide	9%

This patient is clearly septic from pneumonia. This has tipped the patient into atrial fibrillation (AF) given there is no previous history of atrial fibrillation. It is important to treat the sepsis as the cause of the AF. To do this it is imperative to give IV fluids and IV antibiotics. If the patients AF does not settle following the resolution of the sepsis then other options would be considered.

Discuss (7) | Improve

Next question

Sepsis

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to an infection. Sepsis is increasingly recognised as an important cause of mortality in the UK and there has been increasing efforts recently to improve the care of patients who present with sepsis.

How sepsis is classified has changed in recent years - the Surviving Sepsis Guidelines were updated in 2017.

The new guidelines recognise the following terms:

- **sepsis**: life-threatening organ dysfunction caused by a dysregulated host response to infection
- **septic shock**: a more severe form sepsis, technically defined as *'in which circulatory, cellular, and metabolic abnormalities are associated with a greater risk of mortality than with sepsis alone'**

The old category of severe sepsis is no longer used.

The term 'systemic inflammatory response syndrome (SIRS)' has also fallen out of favour. Adult patients outside of ICU with suspected infection are identified as being at heightened risk of mortality if they have quickSOFA (qSOFA) score meeting >= 2 of the following criteria: respiratory rate of 22/min or greater, altered mentation, or systolic blood pressure of 100mmHg or less

qSOFA score
Respiratory rate > 22/min
Altered mentation
Systolic blood pressure < 100 mm Hg

Within an ICU setting a full SOFA** score is often used. Details can be found at the bottom of the notes.

Management

NICE released its own guidelines in 2016. These focussed on the risk stratification and management of patients with suspected.

For risk stratification NICE recommend using the following criteria:

Red flag criteria	Amber flag criteria
• Responds only to voice or pain/ unresponsive • Acute confusional state • Systolic B.P <= 90 mmHg (or drop >40 from normal) • Heart rate > 130 per minute • Respiratory rate >= 25 per minute • Needs oxygen to keep SpO2 >=92% • Non-blanching rash, mottled/ ashen/ cyanotic • Not passed urine in last 18 h/ UO < 0.5 ml/kg/hr	• Relatives concerned about mental status • Acute deterioration in functional ability • Immunosuppressed • Trauma/ surgery/ procedure in last 6 weeks • Respiratory rate 21-24 • Systolic B.P 91-100 mmHg • Heart rate 91-130 OR new dysrhythmia • Not passed urine in last 12-18 hours • Temperature < 36°C • Clinical signs of wound, device or skin infection

Red flag criteria	Amber flag criteria
• Lactate >=2 mmol/l • Recent chemotherapy	

Clearly, the underlying cause of the patient's sepsis needs to be identified and treated and the patient supported regardless of the cause or severity. If however any of the red flags are present the 'sepsis six' should be started straight away:

- 1. Administer oxygen: Aim to keep saturations > 94% (88-92% if at risk of CO2 retention e.g. COPD)
- 2. Take blood cultures
- 3. Give broad-spectrum antibiotics
- 4. Give intravenous fluid challenges
  - NICE recommend a bolus of 500ml crystalloid over less than 15 minutes
- 5. Measure serum lactate
- 6. Measure accurate hourly urine output

*these patients can be clinically identified by a vasopressor requirement to maintain a MAP ≥ 65mmHg and serum lactate >2mmol/L in the absence of hypovolemia

**To help identify and categorise patients the Sequential (Sepsis-Related) Organ Failure Assessment Score (SOFA) is increasingly used. The score grades abnormality by organ system and accounts for clinical interventions. However, laboratory variables, namely, PaO_2, platelet count, creatinine level, and bilirubin level, are needed for full computation.

SOFA Score

System	Score 0	Score 1	Score 2	Score 3	Score 4
PaO_2 /Fl O_2	>400	<400	<300	<200	<100
Platelets x10^3 microlitres	>150	<150	<100	<50	<20
Bilirubin µmol/L	20	20-32	33-101	102-204	>204
Cardiovascular	MAP >70mmHg	MAP 70mmHg	Dopamine <5 or dobutamine (any dose)	Dopamine 5.1-15 or epinephrine 0.1 or norepinephrine 0.1	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1
GCS	15	13-14	10-12	6-9	<6

System	Score 0	Score 1	Score 2	Score 3	Score 4
Creatinine µmol/L	<110	110-170	171-299	300-440	>440
Urine output ml/day	>500	>500	>500	<500	<200

A SOFA score of 2 or more reflects an overall mortality risk of approximately 10% in a general hospital population with suspected infection. Even patients presenting with modest dysfunction can deteriorate further, emphasizing the seriousness of this condition and the need for prompt and appropriate intervention, if not already being instituted.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 4 5

Sepsis Definitions and Diagnosis

European Society of Intensive Care Medicine 2 3

Sepsis-3

Sepsis Trust 0 1

Sepsis clinical toolkit

Journal of American Medical Association

The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)

4 4

NICE

2016 Sepsis: recognition, diagnosis and early management

8 7

Royal College of Physicians

Sepsis: recognition and treatment

3 3

Royal College of Physicians

2011 Sepsis: an update for physicians

4 3

Surviving Sepsis Campaign

Guidelines

6 3

Core EM

Surviving Sepsis 2017 Guidelines Overview

1 0

Suggest link

Report broken link

Media



An introduction to the SEPSIS pathway (detect it early and treat it early)

Armando Hasudungan - YouTube

2 0



Understanding Sepsis (Sepsis Explained Clearly)

Rhesus Medicine - YouTube

0 0

Report broken media

Score: **16.7%**

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Back to top

A 46-year-old woman presents with a 4 week history of cough. This is non-productive and not associated with dyspnoea or chest pain. The symptoms were preceded by a 'head cold'. Her past medical history includes asthma as a child and smoking. Respiratory examination reveals a clear chest with no added sounds. Oxygen saturations are 98% on room air. Temperature is 36.7ºC and heart rate 72/min.

A chest x-ray is requested as she is a smoker:



© Image used on license from Radiopaedia

What is the most likely explanation for the changes on the x-ray?

- Healed varicella pneumonia
- Idiopathic pulmonary fibrosis
- Cryptogenic organizing pneumonia
- Tuberculosis

Sarcoidosis

Submit answer

Reference ranges

Score: **16.7%**

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Back to top

A 46-year-old woman presents with a 4 week history of cough. This is non-productive and not associated with dyspnoea or chest pain. The symptoms were preceded by a 'head cold'. Her past medical history includes asthma as a child and smoking. Respiratory examination reveals a clear chest with no added sounds. Oxygen saturations are 98% on room air. Temperature is 36.7ºC and heart rate 72/min.

A chest x-ray is requested as she is a smoker:



© Image used on license from Radiopaedia

What is the most likely explanation for the changes on the x-ray?

Healed varicella pneumonia	55%
Idiopathic pulmonary fibrosis	4%
Cryptogenic organizing pneumonia	18%
Tuberculosis	11%

Sarcoidosis 13%

The chest x-ray shows miliary opacities secondary to healed varicella pneumonia. Multiple tiny calcific miliary opacities noted throughout both lungs. These are of uniform size and dense suggesting calcification. There is no focal lung parenchymal mass or cavitating lesion seen.The appearances are characteristic for healed varicella pneumonia.

Discuss (8) Improve

Next question

Chickenpox

Chickenpox is caused by primary infection with varicella zoster virus. Shingles is a reactivation of the dormant virus in dorsal root ganglion

Chickenpox is highly infectious

- spread via the respiratory route
- can be caught from someone with shingles
- infectivity = 4 days before rash, until 5 days after the rash first appeared*
- incubation period = 10-21 days

Clinical features (tend to be more severe in older children/adults)

- fever initially
- itchy, rash starting on head/trunk before spreading. Initially macular then papular then vesicular
- systemic upset is usually mild

Management is supportive

- keep cool, trim nails
- calamine lotion
- school exclusion: NICE state the following: *Advise that the most infectious period is 1-2 days before the rash appears, but infectivity continues until all the lesions are dry and have crusted over (usually about 5 days after the onset of the rash).*
- immunocompromised patients and newborns with peripartum exposure should receive varicella zoster immunoglobulin (VZIG). If chickenpox develops then IV aciclovir should be considered

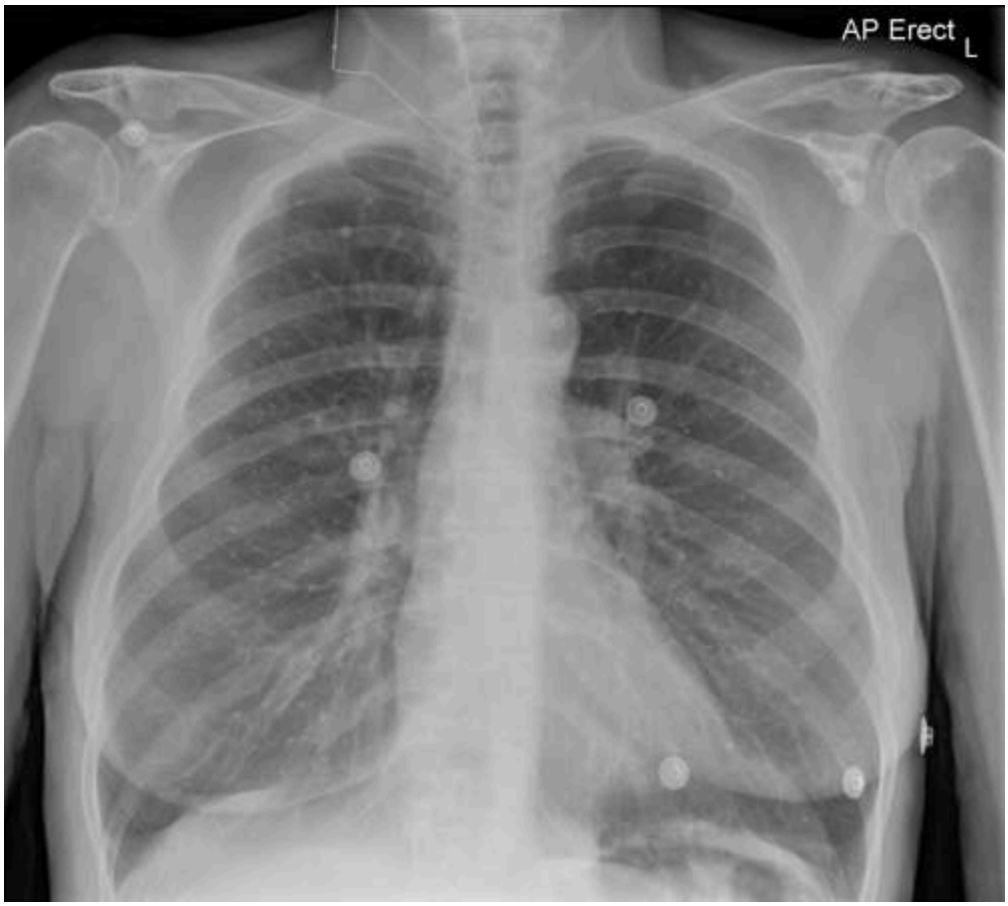
A common complication is secondary bacterial infection of the lesions

- NSAIDs may increase this risk

- whilst this commonly may manifest as a single infected lesion/small area of cellulitis, in a small number of patients invasive group A streptococcal soft tissue infections may occur resulting in necrotizing fasciitis

Rare complications include

- pneumonia
- encephalitis (cerebellar involvement may be seen)
- disseminated haemorrhagic chickenpox
- arthritis, nephritis and pancreatitis may very rarely be seen



© Image used on license from Radiopaedia

Chest x-ray showing miliary opacities secondary to healed varicella pneumonia. Multiple tiny calcific miliary opacities noted throughout both lungs. These are of uniform size and dense suggesting calcification. There is no focal lung parenchymal mass or cavitating lesion seen. The appearances are characteristic for healed varicella pneumonia.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Greenbook 8 6

Varicella

Public Health England 8 6

Chapter 9: managing specific infectious diseases

Suggest link Report broken link

Media



Varicella zoster virus

Osmosis - YouTube 6 0

Report broken media

Score: **16.7%**

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Back to top

A 26-year-old pregnant women is seen on the acute medical unit. She is 24 weeks pregnant with her first child and has been admitted by her GP after feeling generally unwell with 'flu like symptoms and developing jaundiced sclera. Her stay in Bangladesh lasted 2 weeks during which time she visited family. Prior to travelling she was vaccinated against hepatitis A.

On examination her sclera are visibly jaundiced. Blood pressure is 108/60 mmHg, temperature 38.1ºC and pulse 96/min. She is slightly tender in the right upper quadrant of the abdomen. Bloods show the following:

Bilirubin	72µmol/l
ALP	252 u/l
ALT	342 u/l
γGT	286 u/l
Albumin	38 g/l
CRP	154 mg/l

What is the most likely diagnosis?

- Dengue fever
- Hepatitis E
- Hepatitis A
- Intrahepatic cholestasis of pregnancy
- Acute fatty liver

Submit answer

Reference ranges

Score: **16.7%**

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Bilirubin	72µmol/l
ALP	252 u/l
ALT	342 u/l
γGT	286 u/l
Albumin	38 g/l
CRP	154 mg/l

What is the most likely diagnosis?

Dengue fever	8%
Hepatitis E	75%
Hepatitis A	6%
Intrahepatic cholestasis of pregnancy	7%
Acute fatty liver	4%

Hepatitis E is more likely to cause severe disease in pregnant women. Hepatitis A is unlikely given her previous immunisation against this. There are no other features to support the remaining three distractors.

Discuss (2) | Improve

Next question >

Hepatitis E

Overview

- RNA hepevirus
- spread by the faecal-oral route
- incubation period: 3-8 weeks
- common in Central and South-East Asia, North and West Africa, and in Mexico
- causes a similar disease to hepatitis A, but carries a significant mortality (about 20%) during pregnancy
- does not cause chronic disease or an increased risk of hepatocellular cancer
- a vaccine is currently in development, but is not yet in widespread use

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Centers for Disease Control and Prevention

3 1

Hepatitis E review

Suggest link Report broken link

Media



Viral hepatitis

Osmosis - YouTube 1 0



Hepatitis

Podmedics - YouTube 0 1

Report broken media

Score: **16.7%**

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Back to top

A 24-year-old recent immigrant from Albania presents to the emergency department with fever, headache and malaise. Over the past 24 hours he has also developed bilateral pain and swelling at the angle of the jaw, which is made worse by talking or chewing. On examination his pulse is 90/min, temperature 38.4°C and bilateral palpable, tender parotid glands are noted.

Given the likely diagnosis, which one of the following complications is he most likely to develop?

- Orchitis ×
- Pancreatitis ×
- Encephalitis ×
- Myocarditis ×
- Pneumonia ×

Submit answer

Reference ranges

Score: **16.7%**

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Back to top

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Given the likely diagnosis, which one of the following complications is he most likely to develop?

Orchitis	77%
Pancreatitis	5%
Encephalitis	6%
Myocarditis	5%
Pneumonia	7%

Orchitis is the most common complication of mumps in post-pubertal males.

There is a link between mumps and pancreatitis (the 'M' in GET SMASHED) but this is much less common than orchitis.

Discuss (2) | Improve

Next question

Mumps

Mumps is a caused by RNA paramyxovirus and tends to occur in winter and spring

Spread

- by droplets
- respiratory tract epithelial cells → parotid glands → other tissues
- infective 7 days before and 9 days after parotid swelling starts
- incubation period = 14-21 days

Clinical features

- fever
- malaise, muscular pain
- parotitis ('earache', 'pain on eating'): unilateral initially then becomes bilateral in 70%

Prevention
- MMR vaccine: the efficacy is around 80%

Management
- rest
- paracetamol for high fever/discomfort
- notifiable disease

Complications
- orchitis - uncommon in pre-pubertal males but occurs in around 25-35% of post-pubertal males. Typically occurs four or five days after the start of parotitis
- hearing loss - usually unilateral and transient
- meningoencephalitis
- pancreatitis



Next question

Textbooks

High-yield textbook

Extended textbook

Media



Mumps

Osmosis - YouTube 2 0

Report broken media

Score: **16.7%**

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A 41-year-old man with poorly controlled type 1 diabetes mellitus presents with a nodular lesion on the right-side of his face around the angle of the jaw. One month ago he had a tooth extraction at the dentist. The nodule is around 2 cm in diameter, raised and purple-red in colour. On examination a sinus tract is seen in the middle of the nodule which is draining a blood-stained fluid.

Microscopy of the discharge shows microscopic yellow granules.

What is the most likely causative organism?

- *Actinomyces israelii* ×
- *Tunga penetrans* ×
- *Klebsiella* ×
- *Pseudomonas aeruginosa* ×
- *Escherichia coli* ×

Submit answer

Reference ranges

Score: **16.7%**

1 ×
2 ×
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Microscopy of the discharge shows microscopic yellow granules.

What is the most likely causative organism?

Actinomyces israelii	66%
Tunga penetrans	17%
Klebsiella	5%
Pseudomonas aeruginosa	11%
Escherichia coli	1%

The correct answer is ***Actinomyces israelii***. The patient's presentation of a nodular lesion with a sinus tract draining blood-stained fluid and the presence of microscopic yellow granules in the discharge are characteristic features of actinomycosis, which is most commonly caused by *Actinomyces israelii*. Actinomycosis is an indolent, slowly progressive infection that often occurs following dental procedures or oral trauma. The presence of microscopic yellow granules, also known as 'sulfur granules,' further supports this diagnosis.

Tunga penetrans, also known as the chigoe flea, causes tungiasis, which typically presents with pruritic papules or nodules on the feet. This clinical presentation does not match the patient's facial lesion and history of tooth extraction.

Klebsiella species are gram-negative bacteria that can cause a variety of infections, including pneumonia, urinary tract infections, and soft tissue infections. However, these infections do not typically present with nodular lesions and sinus tracts containing sulfur granules.

Pseudomonas aeruginosa is another gram-negative bacterium that can cause various infections such as skin and soft tissue infections, particularly in immunocompromised individuals. Although *Pseudomonas aeruginosa* can cause skin lesions, it does not produce sulfur granules nor form sinus tracts like those seen in actinomycosis.

Finally, ***Escherichia coli*** is a gram-negative bacterium commonly associated with gastrointestinal and urinary tract infections. It can occasionally cause skin and soft tissue infections but would not present with the characteristic nodular lesion containing sulfur granules seen in this patient.

Discuss (2) | Improve

Next question >

Actinomyces and *Nocardia*

Both *Actinomyces* and *Nocardia* are Gram-positive rods that form fungus-like branched networks of hyphae-like filaments.

Actinomyces israelii

Basics

- chronic, progressive granulomatous disease caused by filamentous Gram-positive anaerobic bacteria from the *Actinomycetaceae* family.
- typically causes oral/facial abscesses with sulphur granules in sinus tracts
- may also cause an abdominal mass e.g. in the right iliac fossa

Actinomyces are commensal bacteria that become pathogenic when a mucosal barrier is breached.

The disease most commonly occurs in the head and neck, although it may also occur in the abdominal cavity and in the thorax.

The mass will often enlarge across tissue planes with the formation of multiple sinus tracts.

Abdominopelvic actinomycosis occurs most frequently in individuals that have had appendicitis (65%).

Pathology

- On histological examination Gram-positive organisms and evidence of sulphur granules.
- Sulphur granules are colonies of organisms that appear as round or oval basophilic masses.
- They are also seen in other conditions such as nocardiosis

Treatment

- Long-term antibiotic therapy usually with penicillin
- Surgical resection is indicated for extensive necrotic tissue, non-healing sinus tracts, abscesses or where biopsy is needed to exclude malignancy.

References
Wong V, Turmezei T and Weston V. Actinomycosis. *BMJ* 2011;343d6099.

Nocardia

Basics

- typically causes pneumonia in immunocompromised patients
- may also cause brain abscesses

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 55-year-old man attends after puncturing the skin of his left hand with a small garden fork whilst weeding. On removing the temporary bandage there is no active bleeding but a puncture wound is noted. He has tried to clean the wound but there remains some soil in situ. He is unsure of his tetanus vaccination history but thinks he 'has never not had a vaccine he's been offered'. You can find no record on his tetanus vaccine history on his medical notes. What is the most appropriate course of action?

- Tetanus immunoglobulin
- Tetanus vaccine booster
- Tetanus vaccine booster + tetanus immunoglobulin
- Oral co-amoxiclav
- Tetanus vaccine booster + oral co-amoxiclav

Submit answer

Reference ranges

Score: **16.7%**

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A 55-year-old man attends after puncturing the skin of his left hand with a small garden fork whilst weeding. On removing the temporary bandage there is no active bleeding but a puncture wound is noted. He has tried to clean the wound but there remains some soil in situ. He is unsure of his tetanus vaccination history but thinks he 'has never not had a vaccine he's been offered'. You can find no record on his tetanus vaccine history on his medical notes. What is the most appropriate course of action?

Tetanus immunoglobulin	6%
Tetanus vaccine booster	10%
Tetanus vaccine booster + tetanus immunoglobulin	75%
Oral co-amoxiclav	1%
Tetanus vaccine booster + oral co-amoxiclav	9%

This is a high-risk wound (soil contamination which may contain tetanus spores) in a patient without a clear history of tetanus vaccination. He therefore requires both a tetanus vaccine booster and tetanus immunoglobulin.

Discuss (3) | Improve

Next question

Tetanus: vaccination

The tetanus vaccine is a cell-free purified toxin that is normally given as part of a combined vaccine.

Tetanus vaccine is currently given in the UK as part of the routine immunisation schedule at:

- 2 months
- 3 months
- 4 months
- 3-5 years
- 13-18 years

This, therefore, provides 5 doses of tetanus-containing vaccine. Five doses is now considered to provide adequate long-term protection against tetanus.

Management of wounds

The Greenbook contains the full criteria. The information below is only an abbreviated summary:

The first step is to classify the wound:

Clean wound	Tetanus prone wound	High-risk tetanus prone wound
Wounds less than 6 hours old, non-penetrating with negligible tissue damage	• puncture-type injuries acquired in a contaminated environment e.g. gardening injuries • wounds containing foreign bodies • compound fractures • wounds or burns with systemic sepsis • certain animal bites and scratches	• heavy contamination with material likely to contain tetanus spores e.g. soil, manure • wounds or burns that show extensive devitalised tissue • wounds or burns that require surgical intervention

Patient has had a full course of tetanus vaccines, with the last dose < 10 years ago

- no vaccine nor tetanus immunoglobulin is required, regardless of the wound severity

Patient has had a full course of tetanus vaccines, with the last dose > 10 years ago

- if tetanus prone wound: reinforcing dose of vaccine
- high-risk wounds (e.g. compound fractures, delayed surgical intervention, significant degree of devitalised tissue): reinforcing dose of vaccine + tetanus immunoglobulin

If vaccination history is incomplete or unknown

- reinforcing dose of vaccine, regardless of the wound severity
- for tetanus prone and high-risk wounds: reinforcing dose of vaccine + tetanus immunoglobulin

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Department of Health

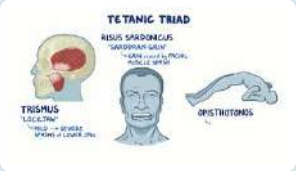
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Tetanus guidelines

Suggest link

Report broken link

Media



Clostridium tetani (tetanus)

Osmosis - YouTube 0 2

Report broken media

Score: **16.7%**

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Back to top

A 52-year-old male presents with a cough, fatigue, fever, and shortness of breath. He had been feeling unwell for around 6 months with recurrent colds that he couldn't seem to shake off. Over this period he had lost around one and a half stone in weight. He attributed this to a poor appetite. His only past medical history was asthma. He had been a widower for 7 years and was a keen wildlife photographer which had allowed him to travel the world. His last trip had been to Africa 2 years ago.

On examination he had a temperature of 38.2ºC, heart rate is 102/min and regular, blood pressure 110/70 mmHg and saturations 92% air. He had fine bibasal crackles on chest auscultation. Heart sounds were normal. Arterial blood gas showed a type 1 respiratory failure. Chest x-ray showed bilateral pulmonary infiltrates. He was treated with intravenous antibiotics. After two days of admission, his condition worsened and his type 1 respiratory failure worsened and he was given continuous positive airway pressure (CPAP). The beta glucan test came back negative. Urinary antigens were negative. The patient remarked that he had noted that his vision in his right eye was blurred and he had noted flashing lights and spots in the same eye.

What is the most likely diagnosis?

- Pneumocystis pneumonia
- CMV pneumonia
- Fungal pneumonia
- Legionella pneumonia
- Asthma exacerbation

Submit answer

Reference ranges

Score: **16.7%**

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What is the most likely diagnosis?

Pneumocystis pneumonia	13%
CMV pneumonia	78%
Fungal pneumonia	6%
Legionella pneumonia	3%
Asthma exacerbation	0%

This gentleman has CMV pneumonia and then developed CMV retinitis on the background of an undiagnosed HIV infection.

CMV infection is typically unnoticed in healthy people but can be life-threatening for those who are immunocompromised, which would include transplant patients, cancer patients, dialysis patients, HIV-infected individuals and those on immunosuppressants.

CMV pneumonia often presents with a cough, fever, malaise, poor appetite, myalgia, shortness of breath and night sweats. CMV can be detected using serology. The virus can also be cultured from urine, throat swabs, bronchial lavages and tissue samples to detect active infection. PCR testing can also be done and this can help monitor viral load.

Ganciclovir or valganciclovir are treatments for CMV infection. Foscarnet or cidofovir are alternatives when patients are resistant to ganciclovir. Foscarnet can cause nephrotoxicity and electrolyte disturbance.

CMV retinitis often presents with blurred vision, floaters, flashing lights, eye pain, photophobia and can lead to blindness. CMV retinitis most commonly develops when the CD4 count is less than 50cells/mL. However, it can occur in 20-30% when then CD4 count is less than 100 cells/mL.

Discuss (9) | Improve

Next question >

Cytomegalovirus

Cytomegalovirus (CMV) is one of the herpes viruses. It is thought that around 50% of people have been exposed to the CMV virus although it only usually causes disease in the immunocompromised, for example people with HIV or those on immunosuppressants following organ transplantation.

Pathophysiology

- infected cells have a 'Owl's eye' appearance due to intranuclear inclusion bodies

Patterns of disease

Congenital CMV infection

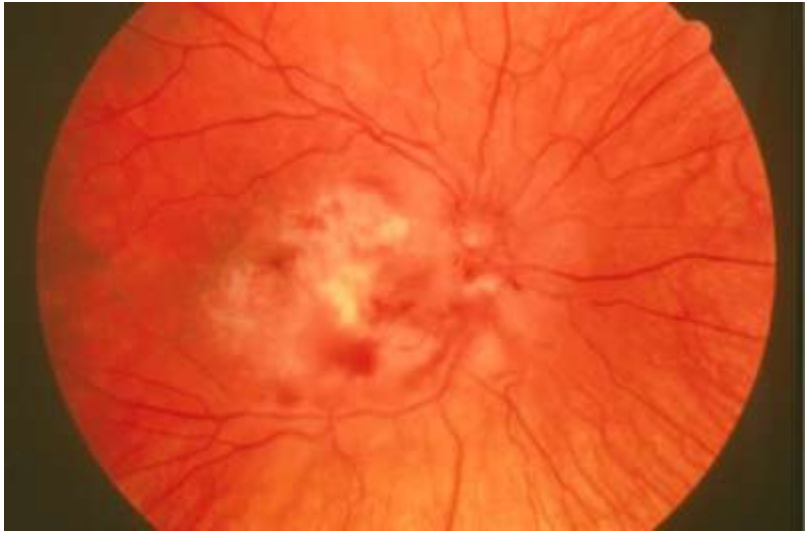
- features include growth retardation, pinpoint petechial 'blueberry muffin' skin lesions, microcephaly, sensorineural deafness, encephalitiis (seizures) and hepatosplenomegaly

CMV mononucleosis

- infectious mononucelosis-like illness
- may develop in immunocompetent individuals

CMV retinitis

- common in HIV patients with a low CD4 count (< 50)
- presents with visual impairment e.g. 'blurred vision'. Fundoscopy shows retinal haemorrhages and necrosis, often called 'pizza' retina
- IV ganciclovir is the treatment of choice



Fundus photograph showing CMV retinitis. Credit: National Eye Institute, National Institutes of Health

CMV encephalopathy

- seen in patients with HIV who have low CD4 counts

CMV pneumonitis

CMV colitis

123

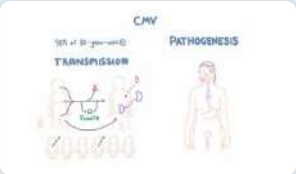
Next question

Textbooks

High-yield textbook

Extended textbook

Media



CMV - Mono, Congenital Infection, Immunocompromised Hosts

macrophage - YouTube 4 0



CMV infection after transplant

Osmosis - YouTube 4 2

Report broken media

Score: **16.7%**

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A 32-year-old woman who has received a renal transplant from an unrelated donor some 4 weeks earlier, comes to the clinic complaining of fevers, arthralgia, abdominal pain and diarrhoea. She had initially made a good recovery after her transplant. Testing for human herpes virus 5 is positive and there has been a rise in CRP and serum creatinine, (values are shown below).

Hb	10.2 g/l	Na^+	138 mmol/l
Platelets	203 * 10^9/l	K^+	5.2 mmol/l
WBC	10.9 * 10^9/l	Urea	9.2 mmol/l
Neuts	8.7 * 10^9/l	Creatinine	211 µmol/l
Lymphs	2.0 * 10^9/l	CRP	88 mg/l
Eosin	0.1 * 10^9/l		

Which of the following is the most appropriate intervention?

- IV acyclovir
- IV ganciclovir
- Oral lamivudine
- Oral valganciclovir
- Oral valaciclovir

Submit answer

Reference ranges

Score: **16.7%**

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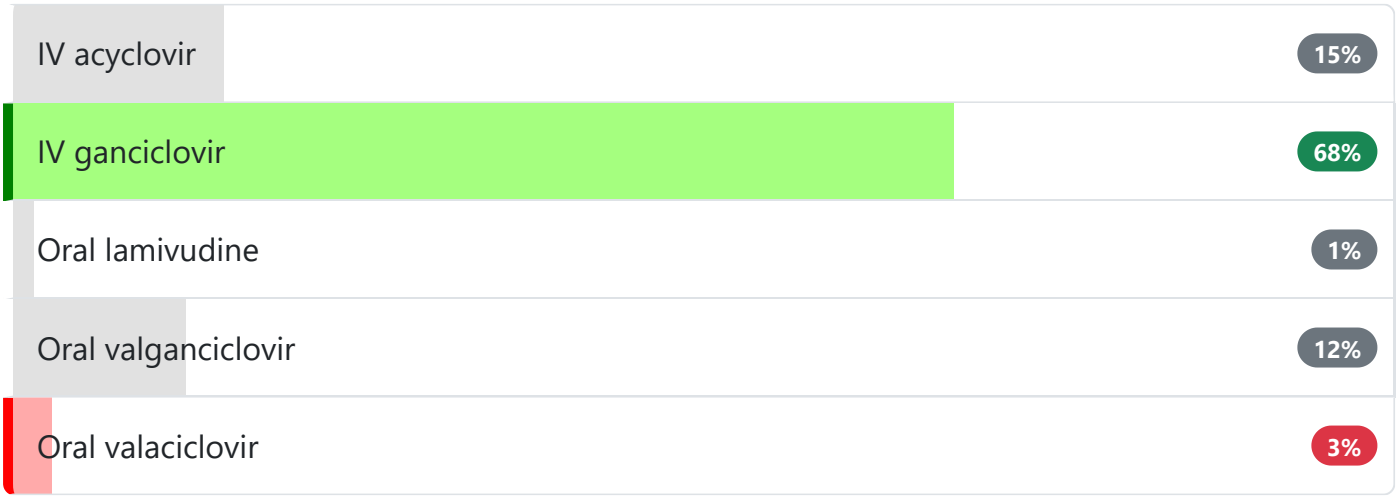
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Eosin	0.1 * 10^9/l		

Which of the following is the most appropriate intervention?



Human herpes virus 5 (HHV 5), is cytomegalovirus (CMV). In patients who are post-transplant, CMV infections can occur from reactivation of latent infection, infection via the transplanted organ, or because of a new primary infection. Treatment is with IV ganciclovir, and where there is ganciclovir resistance, foscarnet or cidofovir are alternative options. Without treatment, further deterioration in renal function is the likely outcome.

IV acyclovir is the usual treatment option for herpes simplex or herpes zoster infection and oral valaciclovir is an alternative option to oral acyclovir for the long term treatment of herpes simplex. Lamivudine is used in the treatment of HIV/AIDs and hepatitis B infection. Valganciclovir is an alternative oral option to IV ganciclovir with potential use for CMV prophylaxis.

Next question

Cytomegalovirus

Cytomegalovirus (CMV) is one of the herpes viruses. It is thought that around 50% of people have been exposed to the CMV virus although it only usually causes disease in the immunocompromised, for example people with HIV or those on immunosuppressants following organ transplantation.

Pathophysiology

- infected cells have a 'Owl's eye' appearance due to intranuclear inclusion bodies

Patterns of disease

Congenital CMV infection

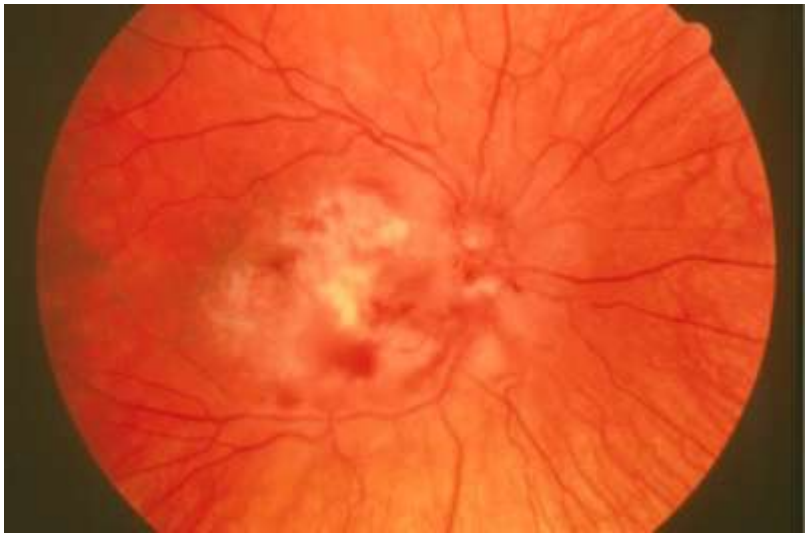
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- presents with visual impairment e.g. 'blurred vision'. Fundoscopy shows retinal haemorrhages and necrosis, often called 'pizza' retina
- IV ganciclovir is the treatment of choice



Fundus photograph showing CMV retinitis. Credit: National Eye Institute, National Institutes of Health

CMV encephalopathy

- seen in patients with HIV who have low CD4 counts

CMV pneumonitis

CMV colitis

AI + Q 123

Next question >

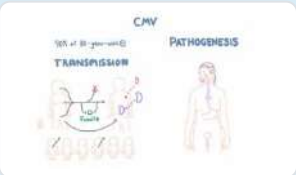
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Textbooks

High-yield textbook

Extended textbook

Media



CMV - Mono, Congenital Infection, Immunocompromised Hosts

macrophage - YouTube 4 0



CMV infection after transplant

Osmosis - YouTube 4 2

Report broken media

Score: **16.7%**

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A 98-year-old lady presents with a fever and confusion and on initial review is started on broad spectrum antibiotics. Despite five days of therapy, her temperature is 38.9°C and her clinical condition seems to have deteriorated. She has no relevant past medical history. The diagnosis of urinary tract infection (UTI) is confirmed when urine cultures confirm an extended spectrum B-lactamase (ESBL) producing *Escherichia coli* (E. coli).

Following the failure to respond to the first-line agent, which antibiotic is most likely to be effective?

- ○ Trimethoprim ✗
- ○ Tetracycline ✗
- ○ Amoxicillin ✗
- ○ Cefalexin ✗
- ○ Ertapenem ✗

Submit answer

Reference ranges

Score: **16.7%**

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Following the failure to respond to the first-line agent, which antibiotic is most likely to be effective?

Trimethoprim	9%
Tetracycline	4%
Amoxicillin	1%
Cefalexin	7%
Ertapenem	**79%**

Extended spectrum B-lactamase (ESBL) producing organisms are typically resistant to penicillins and cephalosporins and as such the carbapenem class of antibiotics are typically first-line although nitrofurantoin or fosfomycin are also frequently effective. ESBL producers are most commonly *Escherichia coli* (E. coli) and *Klebsiella* species.

Discuss (2) | Improve

Next question >

Escherichia coli

Escherichia coli is a facultative anaerobic, lactose-fermenting, Gram negative rod which is a normal gut commensal.

E. coli infections lead to a variety of diseases in humans including:

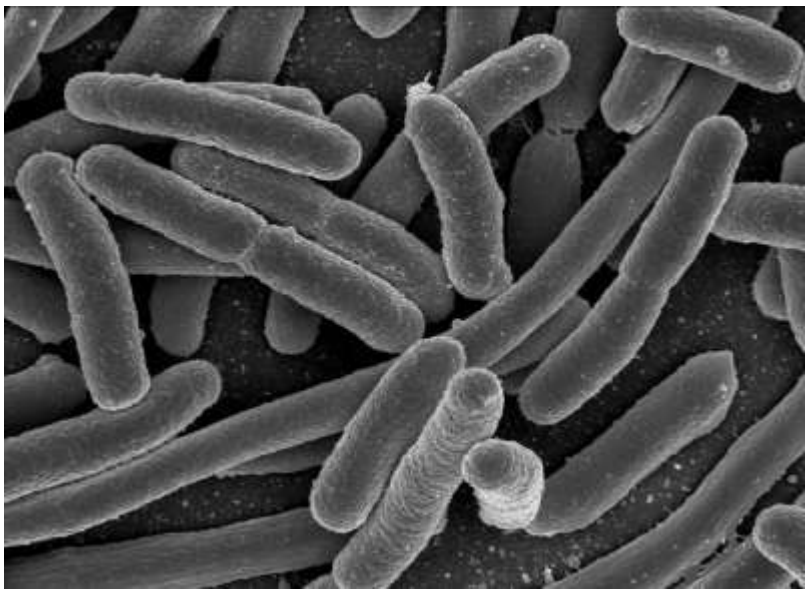
- diarrhoeal illnesses
- UTIs
- neonatal meningitis

Serotypes

E. coli may be classified according to the antigens which may trigger an immune response:

Antigen	Origin	Notes
O	Lipopolysaccharide layer	
K	Capsule	Neonatal meningitis secondary to *E. coli* is usually caused by a serotype that contains the capsular antigen K-1
H	Flagellin	

E. coli O157:H7 is a particular strain associated with severe, haemorrhagic, watery diarrhoea. It has a high mortality rate and can be complicated by haemolytic uraemic syndrome. It is often spread by contaminated ground beef.



Scanning electron micrograph of *Escherichia coli*, grown in culture and adhered to a cover slip. Credit: NIAID

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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You see a 26-year-old Caucasian man with poorly controlled HIV (recent CD4 count of 113 cells/mm^3) with a negative Mantoux test. He was routinely tested after his father (whom he lives with) was diagnosed with active pulmonary disease, but himself shows no features of active disease. On examination, his chest is clear with no overt lymphadenopathy. Blood pressure is 132/91 mmHg, heart rate 85 beats per minute, respiratory rate 14 breaths per minute, oxygen saturation 96% on air and temperature 36.8°C.

What is the next step?

- ○ 5 days oral ciprofloxacin ×
- ○ QuantiFERON-TB (interferon gamma assay) ×
- ○ Induced sputum for AAFB ×
- ○ Monitor and repeat Mantoux in 2 weeks ×
- ○ Rifampicin and isoniazide for 6 months with pyrazinamide and ethambutol for 2 months ×

Submit answer

Reference ranges

Score: **16.7%**

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You see a 26-year-old Caucasian man with poorly controlled HIV (recent CD4 count of 113 cells/mm^3) with a negative Mantoux test. He was routinely tested after his father (whom he lives with) was diagnosed with active pulmonary disease, but himself shows no features of active disease. On examination, his chest is clear with no overt lymphadenopathy. Blood pressure is 132/91 mmHg, heart rate 85 beats per minute, respiratory rate 14 breaths per minute, oxygen saturation 96% on air and temperature 36.8°C.

What is the next step?

5 days oral ciprofloxacin	2%
QuantiFERON-TB (interferon gamma assay)	69%
Induced sputum for AAFB	13%
Monitor and repeat Mantoux in 2 weeks	5%
Rifampicin and isoniazide for 6 months with pyrazinamide and ethambutol for 2 months	11%

In people with HIV and a CD4 count of less than 200 cells/mm^3 an interferon-gamma test should be done alongside the Mantoux as it will be unreliable. If either test is positive a clinical assessment should be performed to exclude active TB and if no active disease found consideration made to treating a latent infection.

Tuberculosis: Clinical diagnosis and management of tuberculosis, and measures for its prevention and control. NICE guidelines (CG117) March 2011

Discuss (7) | Improve

Next question >

Tuberculosis: types

Tuberculosis (TB) is an infection caused by *Mycobacterium tuberculosis* that most commonly affects the lungs. Understanding the pathophysiology of TB can be difficult - the key is to differentiate between primary and secondary disease.

Primary tuberculosis

A non-immune host who is exposed to *M. tuberculosis* may develop a primary infection of the lungs. A small lung lesion known as a Ghon focus develops. The Ghon focus is composed of tubercle-laden macrophages. The combination of a Ghon focus and hilar lymph nodes is known as a Ghon complex

In immunocompetent people, the initial lesion usually heals by fibrosis. Those who are immunocompromised may develop disseminated disease (miliary tuberculosis).

Secondary (post-primary) tuberculosis

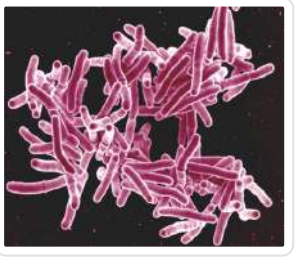
If the host becomes immunocompromised the initial infection may become reactivated. Reactivation generally occurs in the apex of the lungs and may spread locally or to more distant sites. Possible causes of immunocompromise include:

- immunosuppressive drugs including steroids
- HIV
- malnutrition

The lungs remain the most common site for secondary tuberculosis. Extra-pulmonary infection may occur in the following areas:

- central nervous system (tuberculous meningitis - the most serious complication)
- vertebral bodies (Pott's disease)
- cervical lymph nodes (scrofuloderma)
- renal
- gastrointestinal tract





123

Next question

B I

Textbooks

High-yield textbook

Extended textbook

Links

NICE

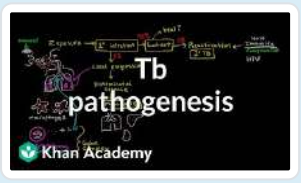
2016 Tuberculosis guidelines

4 4

Suggest link

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Media



TB pathogenesis

Khan Academy - YouTube 1 0



Tuberculosis

Osmosis - YouTube 0 0

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Score: **16.7%**

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A 72-year-old male diabetic is admitted to the hospital via the Emergency Department with severe cellulitis of his right arm. He is hypertensive and also suffers from coronary artery disease.

On examination he is febrile, with a temperature of 38.5°C. His pulse is 112 bpm and his blood pressure is 150/95 mmHg.

His right arm is grossly swollen and tender, with marked erythema and discharge of small amounts of pus.

Lab reports reveal:

Hb	90 g/l
MCV	71 fl
WBC	23 * 10^9/l
Plt	500 * 10^9/l
Urea	9.2 mmol/l
Creatinine	145 µmol/l

Urine examination reveals proteinuria 1+ and glycosuria 2+

Preliminary blood cultures post admission reveal the growth of MRSA.

Which of the following would be the most appropriate antibiotic for this patient?

- IV Vancomycin
- IV Flucloxacillin
- IV Linezolid
- IV Rifampicin
- IV Gentamicin

Submit answer

Reference ranges

Score: **16.7%**

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Creatinine	145 µmol/l

Urine examination reveals proteinuria 1+ and glycosuria 2+

Preliminary blood cultures post admission reveal the growth of MRSA.

Which of the following would be the most appropriate antibiotic for this patient?

- IV Vancomycin — 75%
- IV Flucloxacillin — 4%
- IV Linezolid — 19%
- IV Rifampicin — 0%
- IV Gentamicin — 1%

IV Vancomycin is the treatment of choice for MRSA septicaemia. The renal function is slightly deranged here so the dosing will need to be adjusted accordingly.

MRSA

Methicillin-resistant *Staphylococcus aureus* (MRSA) was one of the first organisms which highlighted the dangers of hospital-acquired infections.

Who should be screened for MRSA?

- all patients awaiting elective admissions (exceptions include day patients having terminations of pregnancy and ophthalmic surgery. Patients admitted to mental health trusts are also excluded)
- from 2011 all emergency admissions will be screened

How should a patient be screened for MRSA?

- nasal swab and skin lesions or wounds
- the swab should be wiped around the inside rim of a patient's nose for 5 seconds
- the microbiology form must be labelled 'MRSA screen'

Suppression of MRSA from a carrier once identified

- nose: mupirocin 2% in white soft paraffin, tds for 5 days
- skin: chlorhexidine gluconate, od for 5 days. Apply all over but particularly to the axilla, groin and perineum

The following antibiotics are commonly used in the treatment of MRSA infections:

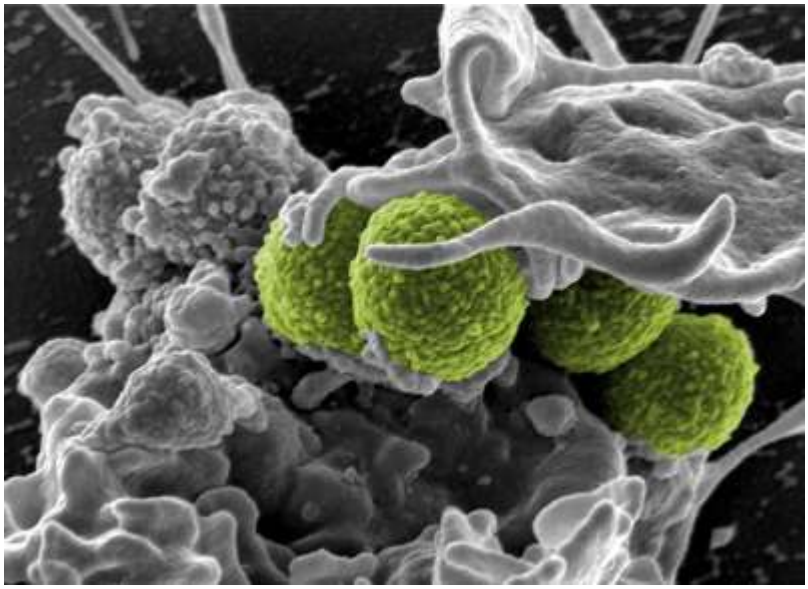
- vancomycin
- teicoplanin
- linezolid

Some strains may be sensitive to the antibiotics listed below but they should not generally be used alone because resistance may develop:

- rifampicin
- macrolides
- tetracyclines
- aminoglycosides
- clindamycin

Relatively new antibiotics such as linezolid, quinupristin/dalfopristin combinations and tigecycline

have activity against MRSA but should be reserved for resistant cases



Interaction of MRSA (green bacteria) with a human white cell. The bacteria shown is strain MRSA252, a leading cause of hospital-associated infections in the United States and United Kingdom. Credit: NIAID

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Health Protection Agency

6 4

2009 MRSA Screening and Suppression guidelines

NICE

5 3

2012 Infection control guidelines

Suggest link Report broken link

Score: **16.7%**

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A 28-year-old patient has presented with upper limb weakness. He is extremely anxious and tells you that this morning he noticed that he had a dry mouth and found it difficult to swallow. His friends also commented that his voice sounded different to normal. Several hours later he began to notice weakness in both of his arms. He has not noticed any weakness in his lower limbs. He has no relevant past medical history or family history of note.
He smokes 10 cigarettes a day, drinks approximately 30 units of alcohol per week and occasionally injects heroin.

His Glasgow Coma Score is 15/15. Neurological examination reveals power 3/5 in the upper limbs, 5/5 in the lower limbs. Biceps and supinator reflexes are absent. Knee and ankle reflexes are normal. His pupils are dilated and sluggish in reaction to light. He is unable to abduct either eye. You notice needle track marks on the patient's forearm and an erythematous wound in the patient's right antecubital fossa.

What is treatment would you give to the patient immediately?

- IV Beta Interferon
- IV immunoglobulin
- Plasma exchange
- Steroids
- Trivalent antitoxin

Submit answer

Reference ranges

Score: **16.7%**

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What is treatment would you give to the patient immediately?

IV Beta Interferon	2%
IV immunoglobulin	17%
Plasma exchange	5%
Steroids	3%
Trivalent antitoxin	73%

This patient has presented with typical features of botulism, which is likely to have been contracted via the patient's wound in this case. He has suffered from a descending weakness, initially affecting his bulbar and pharyngeal muscles, then his upper limbs. If untreated this would progress to involve his respiratory muscles and lower limbs. In contrast, Guillain Barre Syndrome (GBS) presents with an ascending weakness usually involving the lower limbs, then upper limbs then cranial nerves. Both conditions show autonomic features and progress rapidly.

One particularly distressing feature of botulism is that it does not affect GCS.

The immediate treatment of choice would be IV Trivalent antitoxin immediately and general supportive measures.

Next question

Botulism

Clostridium botulinum

- gram positive anaerobic bacillus
- 7 serotypes A-G
- produces botulinum toxin, a neurotoxin which irreversibly blocks the release of acetylcholine
- may result from eating contaminated food (e.g. tinned) or intravenous drug use
- neurotoxin often affects bulbar muscles and autonomic nervous system

Features

- patient usually fully conscious with no sensory disturbance
- flaccid paralysis
- diplopia
- ataxia
- bulbar palsy

Treatment

- botulism antitoxin and supportive care
  - antitoxin is only effective if given early - once toxin has bound its actions cannot be reversed

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.7%**

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A 32-year-old man presents to the emergency department with a three-week history of progressive shortness of breath, dry cough, and fever. He also mentions unintentional weight loss and generalized fatigue in the past two months.

On further questioning, he admits to having had multiple sexual partners in the past year and not using protection consistently.

Observations are as below:

- Temperature 38.9°C (102°F)
- Respiratory rate of 28 breaths/min
- Oxygen saturation of 85% on room air

Arterial blood gases:

Bicarbonate	15 mmol/L	(22 - 29)
PaO2	8.8 kPa	(10.5 - 13.5)
PaCO2	3.8 kPa	(4.5- 6)

Chest examination is unremarkable. The chest x-ray is given below.



© Image used on license from Radiopaedia

What is the most appropriate management in this case?

- ○ Co-trimoxazole ✗
- ○ Co-trimoxazole and steroids ✗
- ○ IV furosemide ✗
- ○ IV pentamidine ✗
- ○ Supportive treatment ✗

Submit answer

Reference ranges

Score: **16.7%**

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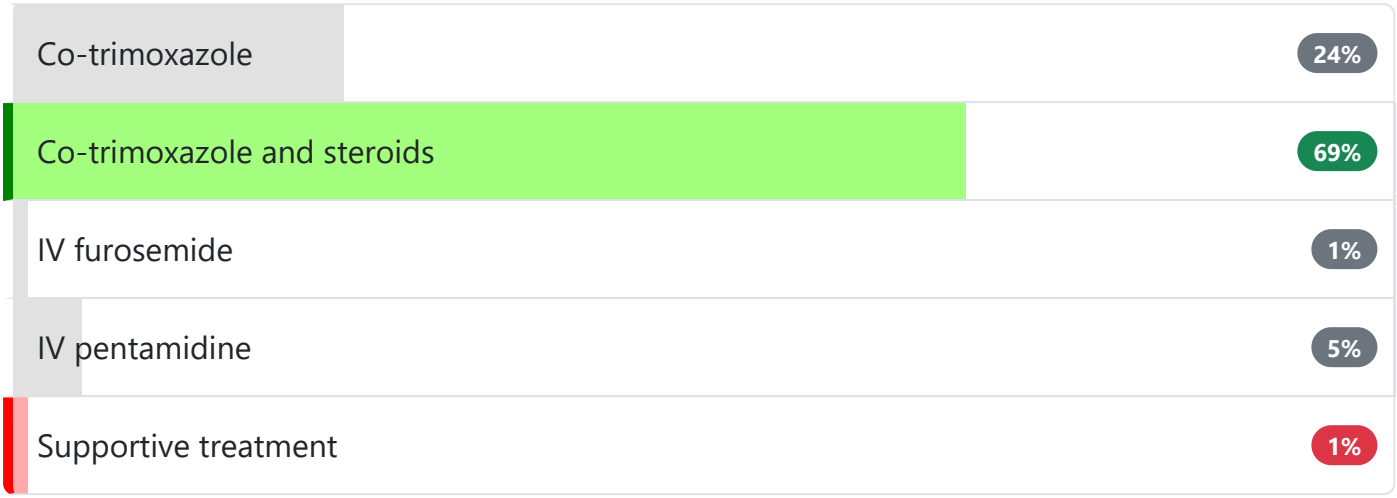
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PaCO2	3.8 kPa	(4.5- 6)

Chest examination is unremarkable. The chest x-ray is given below.



© Image used on license from Radiopaedia

What is the most appropriate management in this case?



Co-trimoxazole and steroids is the correct answer.

The chest x-ray shows increased bibasilar ground glass opacities, combined with the patient's clinical presentation and history of unprotected intercourse, strongly hint at a possible HIV infection complicated by *Pneumocystis jirovecii* pneumonia (also known as PCP). Co-trimoxazole is the first-line treatment for *Pneumocystis jirovecii* pneumonia. The addition of steroids is recommended in hypoxic patients (evidenced here by a PaO2 of 8.8 kPa on air). Steroids have been demonstrated to decrease the risk of respiratory failure by 50% and reduce mortality.

Co-trimoxazole on its own would be inadequate, as the pronounced hypoxia in this patient poses a significant risk of mortality, thus warranting the simultaneous use of steroids.

IV furosemide is not suitable. The imaging changes here are not in keeping with pulmonary oedema, due to peri-hilar distribution of opacification, and lack of other features such as costophrenic angle blunting or fluid in the fissures, and as such diuretic therapy would not be indicated.

IV pentamidine is not the best choice. It can serve as a second-line treatment for *Pneumocystis jirovecii pneumonia* if there are contraindications or adverse reactions to co-trimoxazole but there are no known reasons not to use co-trimoxazole in this case. This patient's marked hypoxia also calls for the administration of steroids.

Supportive treatment on its own isn't adequate. Although vital measures such as oxygen supplementation are necessary for symptom management, they fail to tackle the primary issue - the fungal infection.

Discuss (2) Improve

Next question >

HIV: *Pneumocystis jiroveci* pneumonia

Whilst the organism *Pneumocystis carinii* is now referred to as *Pneumocystis jiroveci*, the term *Pneumocystis carinii* pneumonia (PCP) is still in common use

- *Pneumocystis jiroveci* is an unicellular eukaryote, generally classified as a fungus but some authorities consider it a protozoa
- PCP is the most common opportunistic infection in AIDS
- all patients with a CD4 count < 200/mm^3 should receive PCP prophylaxis

Features

- dyspnoea
- dry cough
- fever
- very few chest signs

Pneumothorax is a common complication of PCP.

Extrapulmonary manifestations are rare (1-2% of cases), may cause

- hepatosplenomegaly
- lymphadenopathy
- choroid lesions

Investigation

- CXR: typically shows bilateral interstitial pulmonary infiltrates but can present with other x-ray findings e.g. lobar consolidation. May be normal
- exercise-induced desaturation
- sputum often fails to show PCP, bronchoalveolar lavage (BAL) often needed to demonstrate PCP (silver stain shows characteristic cysts)





Management

- co-trimoxazole
- IV pentamidine in severe cases
- aerosolized pentamidine is an alternative treatment for *Pneumocystis jiroveci* pneumonia but is less effective with a risk of pneumothorax

- steroids if hypoxic (if pO2 < 9.3kPa then steroids reduce risk of respiratory failure by 50% and death by a third)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF 7 8

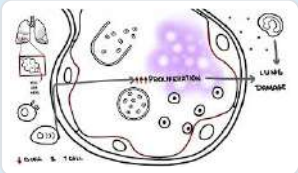
Pneumocystis pneumonia

Patient.info 6 6

Pneumocystis Jirovecii Pneumonia review

Suggest link Report broken link

Media



Pneumocystis jirovecii - Pneumocystis pneumonia

Armando Hasudungan - YouTube 0 0



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

2 1

Report broken media

Score: **16.7%**

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A 25-year-old woman presents to the clinic with a one-month history of persistent, watery diarrhoea that is sometimes difficult to flush but never bloody. She also reports associated bloating and flatulence. Since returning from a trip to South America, she has experienced increasing weakness and an inability to maintain her usual level of physical activity. Over this period, she has lost 10 kg in weight but denies joint pains.

On examination, she is afebrile. Neurological examination shows no abnormalities. Cardiac auscultation reveals normal first (S1) and second (S2) heart sounds without additional sounds or murmurs. There are no skin rashes present.

What is the most likely diagnosis?

Amoebic dysentery	10%
Coeliac disease	5%
Giardiasis	62%
Traveller's diarrhea	2%
Whipple's disease	21%

Ongoing diarrhoea, lethargy, bloating, flatulence, steatorrhoea, weight loss +/- recent travel → ?giardiasis

Important for me Less important

Giardiasis is the correct answer.

Giardiasis should be considered in cases of persistent non-bloody diarrhoea, mainly when it presents as watery and sometimes steatorrhea. Accompanying symptoms may include lethargy, bloating, flatulence, and weight loss. G. lamblia transmission occurs via the fecal-oral route, often through contaminated water or food. The parasite can produce cysts resistant to chlorine and other disinfectants, complicating its eradication from water supplies. Upon ingestion, the cysts excyst within the small intestine and release trophozoites that adhere to the intestinal wall and proliferate.

Amoebic dysentery is incorrect. E.histolytica, the cause of amoebic dysentery, also follows a faecal-oral transmission route, usually via contaminated food or water. Post-ingestion, this parasite may invade the intestinal lining, causing abdominal discomfort and diarrhoea with potentially

bloody stools. Additionally, it can disseminate to other organs, including the liver, lungs, and brain, leading to severe infections which could be life-threatening. However, given the presence of steatorrhea in this scenario, E.histolytica is less likely.

Coeliac disease is a long-term autoimmune disorder primarily affecting the small intestine, where individuals develop intolerance to gluten, which is present in foods such as wheat, rye and barley. Abnormal bowel response to the ingested gluten leads to villous atrophy and subsequent destruction of surface absorption observed in the duodenum and jejunum. Although the symptoms are similar to giardiasis, the relation between symptoms and travelling makes giardiasis more likely. Testing for coeliac disease may be considered part of investigations if the treatment for giardiasis is unsuccessful.

Traveller's diarrhoea is incorrect. *Enterotoxigenic Escherichia coli* (ETEC) is the most common cause of traveller's diarrhoea and is typically associated with watery diarrhoea that may occasionally contain blood rather than steatorrhea. The presence of steatorrhea makes it unlikely.

Whipple's disease caused by *T.whipplei*,—is a rare condition predominantly affecting middle-aged men and can manifest with gastrointestinal symptoms, including steatorrhoea. Nevertheless, the absence of additional signs like joint pain, central nervous system involvement or headaches makes this diagnosis unlikely in this context.

Discuss (1) Improve

Next question >

Giardiasis

Giardiasis is caused by the flagellate protozoan *Giardia lamblia*. It is spread by the faeco-oral route.

Risk factors

- foreign travel
- swimming/drinking water from a river or lake
- male-male sexual contact

Features

- often asymptomatic
- non-bloody diarrhoea
  - steatorrhoea
- bloating, abdominal pain
- lethargy
- flatulence
- weight loss

- malabsorption and lactose intolerance can occur

Investigations

- stool microscopy for trophozoite and cysts: sensitivity of around 65%
- stool antigen detection assay: greater sensitivity and faster turn-around time than conventional stool microscopy methods
- PCR assays are also being developed

Treatment is with metronidazole.

123

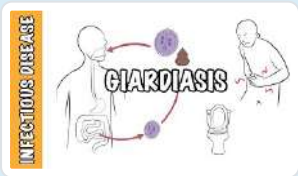
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Giardiasis

Armando Hasudungan - YouTube 2 1

Report broken media

Score: **16.7%**

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A 23-year-old man presents to the emergency department with shortness of breath, fever and a cough. He has had a non-productive cough for two weeks. Before this, he had felt unwell with muscle aches and malaise. He went to his GP and was given amoxicillin but this has made no difference and now he is feeling more breathless and unwell. He has not been outside of the UK in the last year. He has no other medical problems and takes no regular medications. His cough is non-productive and comes in paroxysms which make him concerned.

On examination, there are a few bilateral crepitations, but the examination is otherwise normal. His observations show fever and mild hypoxia. Blood tests show mild anaemia and raised inflammatory markers. A chest X-ray shows bilateral consolidation, but he improves with IV co-amoxiclav and clarithromycin over a few days.

What is the most likely causative organism?

Klebsiella pneumoniae	6%
Legionella pneumophila	12%
Mycoplasma pneumoniae	75%
Chlamydia psittaci	4%
Coxiella burnetii	4%

The correct answer is Mycoplasma pneumoniae. There are several hints in this history and investigations to suggest Mycoplasma pneumoniae as the likely cause. First of all, he is a young and fit patient who describes a non-productive cough preceded by flu-like symptoms and not responding to penicillins. Secondly, he has anaemia which may be due to haemolysis, and finally there is bilateral consolidation on the chest X-ray. Klebsiella pneumoniae is more typical in alcoholics, Legionella pneumophila is associated with a history of foreign travel, Chlamydia psittaci is associated with contact and exposure to birds and finally, Coxiella burnetii is rare and associated with farm animal exposure.

Discuss (3) | Improve

Next question

Mycoplasma pneumoniae

Mycoplasma pneumoniae is a cause of atypical pneumonia which often affects younger patients. It is associated with a number of characteristic complications such as erythema multiforme and cold autoimmune haemolytic anaemia. Epidemics of *Mycoplasma pneumoniae* classically occur every 4 years. It is important to recognise atypical pneumonia as it may not respond to penicillins or cephalosporins due to it lacking a peptidoglycan cell wall.

Features

- the disease typically has a prolonged and gradual onset
- flu-like symptoms classically precede a dry cough
- bilateral consolidation on x-ray
- complications may occur as below

Complications

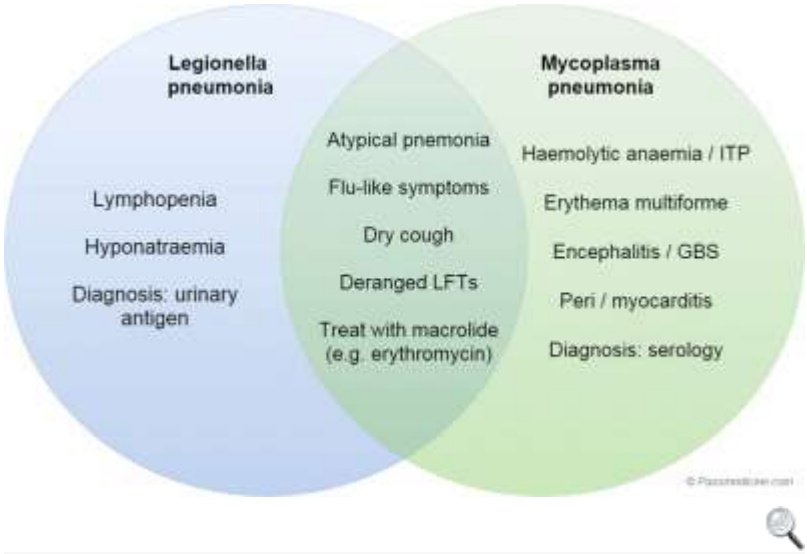
- cold agglutins (IgM): may cause an haemolytic anaemia, thrombocytopenia
- erythema multiforme, erythema nodosum
- meningoencephalitis, Guillain-Barre syndrome and other immune-mediated neurological diseases
- bullous myringitis: painful vesicles on the tympanic membrane
- pericarditis/myocarditis
- gastrointestinal: hepatitis, pancreatitis
- renal: acute glomerulonephritis

Investigations

- diagnosis is generally by Mycoplasma serology
- positive cold agglutination test → peripheral blood smear may show red blood cell agglutination

Management

- doxycycline or a macrolide (e.g. erythromycin/clarithromycin)



Comparison of Legionella and Mycoplasma pneumonia

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

11 6

Erythema multiforme

Suggest link

Report broken link

Media



Mycoplasma pneumoniae

Osmosis - YouTube 1 0

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Back to top

A 35-year-old gentleman presents to the emergency department with worsening shortness of breath on exertion. His symptoms have been progressing for two weeks, and he now finds it difficult to take a full breath. He has no chest pain and has not been coughing but he has been feeling increasingly fatigued. He has a past medical history of HIV. He has been struggling to take his tablets recently. He is unsure of the names of any tablets, but he knows that he is supposed to take one tablet each day at the same time and to take two tablets twice a day on Mondays, Wednesdays, and Fridays. He has no other medical problems.

On examination, auscultation shows normal breath sounds.

Observations:

Saturations	93%
Respiratory rate	22/min
Blood pressure	136/71mmHg
Heart rate	91/min
Temperature	37.6°C

On mobilisation, his saturations reduce to 88%.

Chest X-ray is clear.

What investigation is most likely to be diagnostic?

- CT pulmonary angiogram
- High resolution CT chest
- ECG
- Echocardiogram
- Broncho-alveolar lavage

Submit answer

Reference ranges

Score: **16.7%**

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Observations:

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Respiratory rate	22/min
Blood pressure	136/71mmHg
Heart rate	91/min
Temperature	37.6°C

On mobilisation, his saturations reduce to 88%.

Chest X-ray is clear.

What investigation is most likely to be diagnostic?

CT pulmonary angiogram	4%
High resolution CT chest	18%
ECG	1%
Echocardiogram	3%
Broncho-alveolar lavage	**74%**

The correct answer is broncho-alveolar lavage which is most likely to be able to demonstrate PCP characteristic cysts with silver staining. There are several factors highly suggestive of PCP. This gentleman is poorly compliant with his HIV medication and the other medication regime he is describing is PCP prophylaxis, suggesting a low CD4 count. He also is tachypnoeic with low saturations on mobilising and a clear chest X-ray. All of these factors suggest PCP. In the case of a

productive cough, sputum could be obtained without the need for a broncho-alveolar lavage. Imaging can be suggestive of PCP but is not as sensitive or diagnostic as broncho-alveolar lavage. ECG and echocardiogram have no role in PCP diagnosis.

Source:
Nelson, M., Dh Dockrell, and S. Edwards. 'British HIV Association and British Infection Association Guidelines for the Treatment of Opportunistic Infection in HIV-seropositive Individuals 2011.' HIV Medicine 12 (2011): 1-5.

Discuss (4) | Improve

Next question >

HIV: *Pneumocystis jiroveci* pneumonia

Whilst the organism *Pneumocystis carinii* is now referred to as *Pneumocystis jiroveci*, the term *Pneumocystis carinii* pneumonia (PCP) is still in common use

- *Pneumocystis jiroveci* is an unicellular eukaryote, generally classified as a fungus but some authorities consider it a protozoa
- PCP is the most common opportunistic infection in AIDS
- all patients with a CD4 count < 200/mm^3 should receive PCP prophylaxis

Features

- dyspnoea
- dry cough
- fever
- very few chest signs

Pneumothorax is a common complication of PCP.

Extrapulmonary manifestations are rare (1-2% of cases), may cause

- hepatosplenomegaly
- lymphadenopathy
- choroid lesions

Investigation

- CXR: typically shows bilateral interstitial pulmonary infiltrates but can present with other x-ray findings e.g. lobar consolidation. May be normal
- exercise-induced desaturation
- sputum often fails to show PCP, bronchoalveolar lavage (BAL) often needed to demonstrate PCP (silver stain shows characteristic cysts)





Management

- co-trimoxazole
- IV pentamidine in severe cases
- aerosolized pentamidine is an alternative treatment for *Pneumocystis jiroveci* pneumonia but is less effective with a risk of pneumothorax
- steroids if hypoxic (if pO2 < 9.3kPa then steroids reduce risk of respiratory failure by 50% and death by a third)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF

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Pneumocystis pneumonia

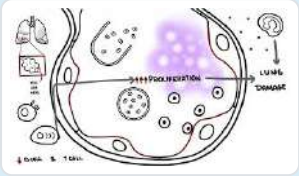
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Pneumocystis Jirovecii Pneumonia review

Suggest link Report broken link

Media



Pneumocystis jirovecii - Pneumocystis pneumonia

Armando Hasudungan - YouTube 0 0



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube 2 1

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Score: **19%**

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A 42-year-old woman presents to the emergency department with a dry cough. Over the last five days, she has been suffering from a worsening dry cough, shortness of breath that is worse on exertion, and she has felt hot. She has a past medical history of HIV which was recently diagnosed, but no other medical problems. She has been started two months ago on antiretroviral treatment and PCP prophylaxis, to which she reports good compliance.

Fundoscopy shows white branches alongside blood vessels. She denies any eye pain or change in her vision.

Observations:

Saturations	93%
Respiratory rate	20/min
Blood pressure	131/70mmHg
Heart rate	89/min
Temperature	38.1°C

Blood tests:

Hb	122 g/l
Platelets	401 * 10^9/l
WBC	10.2 * 10^9/l
Na^+	138 mmol/l
K^+	4.2 mmol/l
Urea	5.1 mmol/l
Creatinine	71 µmol/l
HIV viral load	12,198 copies/ml
CD4 count	70 cells/mm^3

A chest X-ray shows bilateral interstitial infiltrates. A broncho-alveolar lavage is organised but shows no organisms on microscopy, including with silver staining. PCR studies are pending.

What is the most appropriate treatment?

- Prednisolone
- Low-molecular weight heparin

- Tazocin
- IV co-trimoxazole
- Ganciclovir

Submit answer

Reference ranges

Score: **16.7%**

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A chest X-ray shows bilateral interstitial infiltrates. A broncho-alveolar lavage is organised but shows no organisms on microscopy, including with silver staining. PCR studies are pending.

What is the most appropriate treatment?

Prednisolone	8%
Low-molecular weight heparin	1%

Tazocin	1%
IV co-trimoxazole	34%
Ganciclovir	55%

The correct answer is ganciclovir as this patient is likely to have CMV pneumonitis. Prednisolone would be more appropriate during PCP treatment, heparin would be appropriate if a pulmonary embolus was expected, tazocin is a broad-spectrum antibiotic used in many bacterial infection whilst co-trimoxazole is first line treatment for PCP. There are are several factors strongly suggestive of CMV as the cause of infection; fever with desaturation in poorly controlled HIV and evidence of retinitis as well. 'Chest X-ray of bilateral interstitial infiltrates is highly suggestive too. The diagnosis should be confirmed by a biopsy or broncho-alveolar lavage which can use PCR to amplify CMV. The negative silver staining makes PCP less likely as well.

Source:
Nelson, M., Dh Dockrell, and S. Edwards. 'British HIV Association and British Infection Association Guidelines for the Treatment of Opportunistic Infection in HIV-seropositive Individuals 2011.' HIV Medicine 12 (2011): 1-5.

Discuss (8) Improve

Next question

Cytomegalovirus

Cytomegalovirus (CMV) is one of the herpes viruses. It is thought that around 50% of people have been exposed to the CMV virus although it only usually causes disease in the immunocompromised, for example people with HIV or those on immunosuppressants following organ transplantation.

Pathophysiology
- infected cells have a 'Owl's eye' appearance due to intranuclear inclusion bodies

Patterns of disease

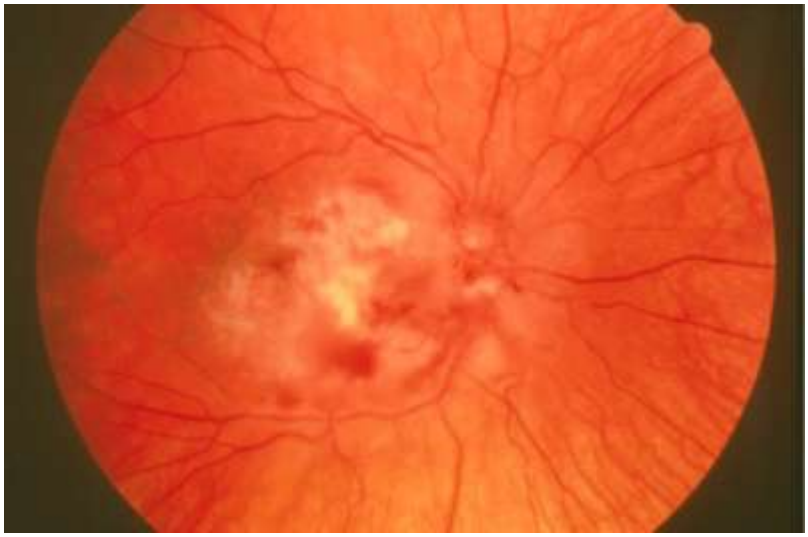
Congenital CMV infection
- features include growth retardation, pinpoint petechial 'blueberry muffin' skin lesions, microcephaly, sensorineural deafness, encephalitiis (seizures) and hepatosplenomegaly

CMV mononucleosis

- infectious mononucelosis-like illness
- may develop in immunocompetent individuals

CMV retinitis

- common in HIV patients with a low CD4 count (< 50)
- presents with visual impairment e.g. 'blurred vision'. Fundoscopy shows retinal haemorrhages and necrosis, often called 'pizza' retina
- IV ganciclovir is the treatment of choice



Fundus photograph showing CMV retinitis. Credit: National Eye Institute, National Institutes of Health

CMV encephalopathy

- seen in patients with HIV who have low CD4 counts

CMV pneumonitis

CMV colitis

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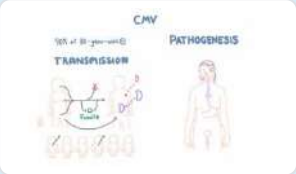
Next question

Textbooks

High-yield textbook

Extended textbook

Media



CMV - Mono, Congenital Infection, Immunocompromised Hosts

macrophage - YouTube 4 0



CMV infection after transplant

Osmosis - YouTube 4 2

Report broken media

Score: **19%**

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Back to top

A 71-year-old gentleman presented to the emergency department with a headache, myalgia and fever. These symptoms all started one day ago. He has a past medical history of hypertension, ischaemic heart disease, type 2 diabetes, congestive cardiac failure and bilateral hip replacement. His medications include amlodipine, ramipril, aspirin, atorvastatin, gliclazide, calcium carbonate with colecalciferol (Adcal-D3) and paracetamol. He denies any allergies. He is concerned that he about influenza as he visited a friend in hospital last week who had been diagnosed with influenza A.

On examination, he appears sweaty and feels hot to touch. His chest is clear on auscultation. He is placed in a side room for observation and viral throat swabs are requested.

Observations:

Oxygen saturation	96% on room air
Respiratory rate	18/min
Blood pressure	129/93mmHg
Heart rate	77/min
Temperature	38.1°C

What is the most appropriate treatment?

- Supportive management only
- Salbutamol
- Zanamivir
- Amantadine
- Oseltamivir

Submit answer

Reference ranges

Score: **16.7%**

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Observations:

Oxygen saturation	96% on room air
Respiratory rate	18/min
Blood pressure	129/93mmHg
Heart rate	77/min
Temperature	38.1°C

What is the most appropriate treatment?

Supportive management only	27%
Salbutamol	1%
Zanamivir	7%
Amantadine	1%
Oseltamivir	**64%**

The correct answer is oseltamivir. This is a patient presenting with symptoms of influenza following recent exposure. The first aspect to consider is if he needs any antiviral drugs. Since he presented within 48 hours of the onset of symptoms and is in an at-risk group because of his diabetes he would warrant antiviral treatment and therefore supportive management is not the most appropriate treatment. Salbutamol is not indicated for flu alone. Zanamivir is second line treatment for influenza, whilst amantadine is no longer recommended for use in the treatment of influenza.

Discuss (10) | Improve

Next question

Influenza

Features

The following are typically seen:

- fever greater than 38ºC
- myalgia
- lethargy
- headache
- rhinitis
- sore throat
- cough
- diarrhoea and vomiting

Management

Consider prescribing antiviral treatment for influenza if all of the following apply:

- 1) The patient is in an at-risk group or is felt to be at risk of developing a serious complication
  - > 65 years old
  - pregnant women
  - chronic disease of respiratory, cardiac, renal, hepatic or neurological nature
  - diabetes
  - immunosuppression
  - morbid obesity.
- 2) There is circulating influenza nationally
  - this would mean typically the winter months
- 3) The patient is able to start treatment within 48 hours from the onset of symptoms

Antivirals for influenza (simplified, please see the CKS link for more details)

- First line: oseltamivir
- Second line: zanamivir

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 0 0

Scenario: Treatment of influenza

Suggest link Report broken link

Media



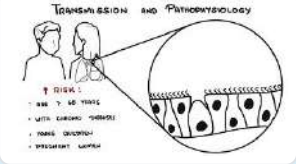
Influenza

Osmosis - YouTube 4 0



Flu - What is it?

Let's Learn About Bugs - YouTube 3 0



Understanding Influenza - The Flu

Armando Hasudungan - YouTube 0 0

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An 82-year-old gentleman inpatient is reviewed on the ward round. He originally presented with diarrhoea on a background of ulcerative colitis flare and is managed with oral and rectal mesalazine. His bowel has settled but he developed a rash around his left antecubital fossa, and he has developed a temperature of 38.2ºC. On examination, his chest sounds clear and his abdomen is soft, but the left antecubital fossa has a tender and warm erythematous rash. Stool results show no growth, abdominal and chest X-ray are clear but a MRSA swab from the nose is positive. What is the most appropriate treatment?

- Mupirocin and chlorhexidine ×
- Benzylpenicillin ×
- Tazocin ×
- Vancomycin ×
- Flucloxacillin ×

Submit answer

Reference ranges

Score: **16.7%**

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Mupirocin and chlorhexidine	26%
Benzylpenicillin	1%
Tazocin	2%
Vancomycin	63%
Flucloxacillin	8%

The correct answer is vancomycin. This is a patient with a new fever and the most obvious cause is cellulitis. Given that the location of the cellulitis is in a location common for cannulation the most likely explanation is iatrogenic cellulitis. This makes MRSA a much higher likelihood and therefore the most appropriate treatment is vancomycin. Mupirocin and chlorhexidine are used to eradicate MRSA when present as commensal organisms. In non-iatrogenic cellulitis benzylpenicillin and flucloxacillin are commonly used.

Discuss (3) | Improve

Next question >

MRSA

Methicillin-resistant *Staphylococcus aureus* (MRSA) was one of the first organisms which highlighted the dangers of hospital-acquired infections.

Who should be screened for MRSA?

- all patients awaiting elective admissions (exceptions include day patients having terminations of pregnancy and ophthalmic surgery. Patients admitted to mental health trusts are also excluded)

- from 2011 all emergency admissions will be screened

How should a patient be screened for MRSA?

- nasal swab and skin lesions or wounds
- the swab should be wiped around the inside rim of a patient's nose for 5 seconds
- the microbiology form must be labelled 'MRSA screen'

Suppression of MRSA from a carrier once identified

- nose: mupirocin 2% in white soft paraffin, tds for 5 days
- skin: chlorhexidine gluconate, od for 5 days. Apply all over but particularly to the axilla, groin and perineum

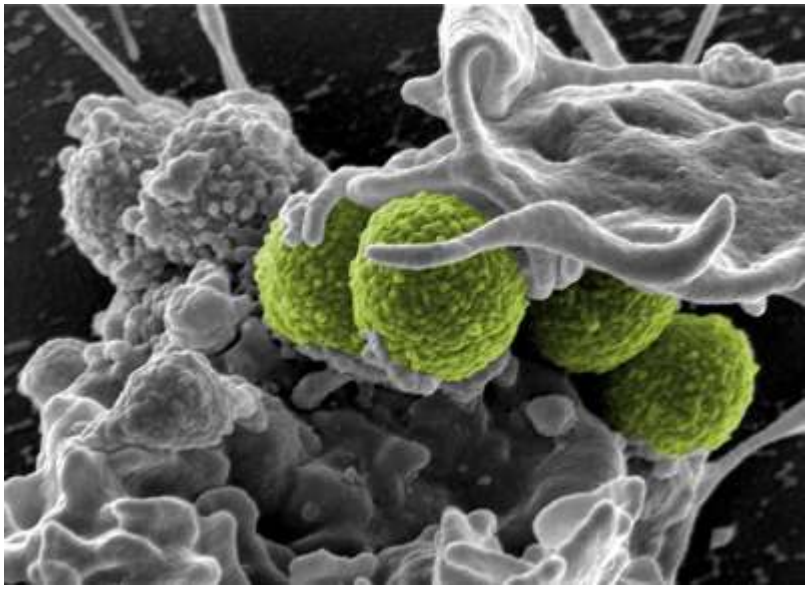
The following antibiotics are commonly used in the treatment of MRSA infections:

- vancomycin
- teicoplanin
- linezolid

Some strains may be sensitive to the antibiotics listed below but they should not generally be used alone because resistance may develop:

- rifampicin
- macrolides
- tetracyclines
- aminoglycosides
- clindamycin

Relatively new antibiotics such as linezolid, quinupristin/dalfopristin combinations and tigecycline have activity against MRSA but should be reserved for resistant cases



Interaction of MRSA (green bacteria) with a human white cell. The bacteria shown is strain MRSA252, a leading cause of

hospital-associated infections in the United States and United Kingdom. Credit: NIAID

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Health Protection Agency 6 4

2009 MRSA Screening and Suppression guidelines

NICE 5 3

2012 Infection control guidelines

Suggest link Report broken link

Score: **19%**

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A 32-year-old gentleman presents to the emergency department with severe nausea, vomiting and diarrhoea. He was recently at a reunion where 18 out of 25 guests have developed similar symptoms shortly afterwards. On examination he appears clinically dehydrated but his vital parameters are all normal. He has no past medical history and otherwise his examination is normal. You suspect norovirus; what is the most appropriate investigation?

- Serum serology ✗
- Faecal or vomitus toxicology ✗
- Faecal or vomitus viral PCR ✗
- Serum toxins ✗
- Serum viral PCR ✗

Submit answer

Reference ranges

Score: **16.7%**

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Serum serology	4%
Faecal or vomitus toxicology	5%
Faecal or vomitus viral PCR	70%
Serum toxins	4%
Serum viral PCR	17%

The correct answer is faecal or vomitus viral PCR which can confirm norovirus infection. Tests can detect antibodies by the ELISA method but these lack sensitivity and specificity and are therefore seldom used in the clinical setting. Norovirus does not make a toxin and therefore there is no such thing as norovirus toxin testing. The virus is not normally present in the bloodstream and therefore serum is likely to be negative for PCR testing. Norovirus is a viral cause of gastroenteritis commonly associated with large outbreaks in winter.

Discuss (3) | Improve

Next question

Gastroenteritis: causes

Gastroenteritis may either occur whilst at home or whilst travelling abroad (travellers' diarrhoea)

Travellers' diarrhoea may be defined as at least 3 loose to watery stools in 24 hours with or without one of more of abdominal cramps, fever, nausea, vomiting or blood in the stool. The most common cause is *Escherichia coli*.

Another pattern of illness is 'acute food poisoning'. This describes the sudden onset of nausea, vomiting and diarrhoea after the ingestion of a toxin. Acute food poisoning is typically caused by

Staphylococcus aureus, *Bacillus cereus* or *Clostridium perfringens*.

Stereotypical histories

Infection	Typical presentation
Escherichia coli	Common amongst travellers Watery stools Abdominal cramps and nausea
Giardiasis	Prolonged, non-bloody diarrhoea
Cholera	Profuse, watery diarrhoea Severe dehydration resulting in weight loss Not common amongst travellers
Shigella	Bloody diarrhoea Vomiting and abdominal pain
Staphylococcus aureus	Severe vomiting Short incubation period
Campylobacter	A flu-like prodrome is usually followed by crampy abdominal pains, fever and diarrhoea which may be bloody May mimic appendicitis Complications include Guillain-Barre syndrome
Bacillus cereus	Two types of illness are seen • vomiting within 6 hours, stereotypically due to rice • diarrhoeal illness occurring after 6 hours
Amoebiasis	Gradual onset bloody diarrhoea, abdominal pain and tenderness which may last for several weeks

Incubation period

- 1-6 hrs: *Staphylococcus aureus*, *Bacillus cereus**
- 12-48 hrs: *Salmonella*, *Escherichia coli*
- 48-72 hrs: *Shigella*, *Campylobacter*
- > 7 days: Giardiasis, Amoebiasis

*vomiting subtype, the diarrhoeal illness has an incubation period of 6-14 hours

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians

16 13

2011 Diarrhoeal disease review

Suggest link

Report broken link

Media



Shigella

ATP - YouTube 5 0



Salmonella

ATP - YouTube 5 0

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A 69-year-old woman with diabetes and rheumatoid arthritis presents to the clinic complaining of malaise. She takes metformin, sulfasalazine and hydroxychloroquine daily and methotrexate once a week. She volunteers at a care home near her home. On examination, she appears frail, and her fingers and toes are deformed. She is alert, afebrile and comfortable at rest. Her pulse rate is 70 beats per minute, BP is 120/80 mmHg, and respiratory rate is 16 breaths per minute. On inspection of her legs, you notice the following:



© Image used on license from DermNet NZ

What is the most appropriate next step in the management of this patient?

- Admit and treat with intravenous antibiotics
- Order for an x-ray of the limb
- Order for blood cultures and review with reports after one week
- Prescribe oral antibiotics and arrange a follow-up visit for the next week
- Prescribe oral prednisolone and arrange a follow-up visit

Submit answer

Reference ranges

Score: **16.7%**

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Back to top

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© Image used on license from DermNet NZ

What is the most appropriate next step in the management of this patient?

Admit and treat with intravenous antibiotics	67%
Order for an x-ray of the limb	5%
Order for blood cultures and review with reports after one week	2%
Prescribe oral antibiotics and arrange a follow-up visit for the next week	24%
Prescribe oral prednisolone and arrange a follow-up visit	2%

The most appropriate next step is to **admit and treat with intravenous antibiotics**. The image provided shows a swollen and erythematous right lower limb. Differential diagnoses for a swollen limb include cellulitis, deep vein thrombosis (DVT), lymphoedema, and fluid overload conditions such as chronic heart failure. Cellulitis should be suspected when there is a history of fever and

malaise, with superficial bullae potentially observed over the affected area. DVT typically presents in patients who have risk factors (such as recent immobilisation, surgery, or pregnancy) with unilateral limb oedema, erythema, and tenderness. Lymphoedema often has an insidious onset and manifests as a slowly progressive swelling of a limb; it is commonly observed in patients post-lymph node dissection proximal to the affected area. Fluid overload states usually present with bilateral pitting pedal oedema without associated warmth or tenderness.

In this case, we are presented with a frail, immunocompromised woman exhibiting symptoms of right leg cellulitis. These characteristics'frailty and an immunocompromised state'are indications for in-patient management according to NICE guidelines on admission criteria for cellulitis. It is important to note that methotrexate is typically withheld for patients who are being hospitalized with severe infections requiring intravenous antibiotic therapy.

Ordering an x-ray of the limb is not correct. While cellulitis is diagnosed clinically, an x-ray may be warranted only if there is strong clinical suspicion of osteomyelitis in the underlying bone; otherwise, it does not play a role in the management of cellulitis itself. Ultrasound (USG), computed tomography (CT), and magnetic resonance imaging (MRI) can help exclude deeper tissue infections such as myositis or detect abscess formation.

Ordering blood cultures and reviewing after one week is inappropriate for this scenario. This patient has an acute infection necessitating immediate antibiotic treatment. Blood cultures are indicated when sepsis is suspected but should not delay empirical antibiotic therapy aimed at preventing infection progression.

Prescribing oral antibiotics and arranging follow-up is incorrect for this patient's presentation. Given her frailty and immunocompromised status, she requires hospital admission for intravenous antibiotic therapy rather than outpatient management with oral antibiotics, which are reserved for Eron class I and II cases of cellulitis.

Prescribing oral prednisolone would be ill-advised here as it could exacerbate the infection by suppressing the immune response instead of treating it. Prednisolone may be indicated in patients experiencing acute rheumatoid arthritis flares but is not applicable to this clinical situation.

Discuss (2) | Improve

Next question >

Cellulitis ★

Cellulitis is a bacterial infection that affects the dermis and the deeper subcutaneous tissues. It is one of the most common conditions seen in the acute medical setting and is usually diagnosed clinically. Cellulitis is most commonly caused by infection with *Streptococcus pyogenes* or less commonly *Staphylcoccus aureus*. The majority of cases resolve with oral antibiotics.

Features

- commonly occurs on the shins
  - usually unilateral - bilateral cellulitis is rare and suggests an alterative diagnosis
- erythema
  - generally reasonably well-defined margins but some cases may present with diffuse erythema
  - blisters and bullae may be seen with more severe disease
- swelling
- systemic upset
  - fever
  - malaise
  - nausea





The diagnosis of cellulitis is clinical. No further investigations are required in primary care. Bloods and blood cultures may be requested if the patient is admitted and septicaemia is suspected.

Criteria for admission

The Eron classification system can be used to guide how we manage patients with cellulitis:

Class	Features
I	There are no signs of systemic toxicity and the person has no uncontrolled co-morbidities
II	The person is either systemically unwell or systemically well but with a co-morbidity (for example peripheral arterial disease, chronic venous insufficiency, or morbid obesity) which may complicate or delay resolution of infection
III	The person has significant systemic upset such as acute confusion, tachycardia, tachypnoea, hypotension, or unstable co-morbidities that may interfere with a response to treatment, or a limb-threatening infection due to vascular compromize
IV	The person has sepsis syndrome or a severe life-threatening infection such as necrotizing fasciitis

They recommend the following that we admit for intravenous antibiotics the following patients:

- Has Eron Class III or Class IV cellulitis.
- Has severe or rapidly deteriorating cellulitis (for example extensive areas of skin).
- Is very young (under 1 year of age) or frail.
- Is immunocompromized.
- Has significant lymphoedema.
- Has facial cellulitis (unless very mild) or periorbital cellulitis.

The following is recommended regarding Eron Class II cellulitis:

> *Admission may not be necessary if the facilities and expertise are available in the community to give intravenous antibiotics and monitor the person - check local guidelines.*

Other patients can be treated with oral antibiotics.

Management

Management is guided by the Eron classification.

Eron Class I

- oral antibiotics NICE
- oral flucloxacillin as first-line treatment for mild/moderate cellulitis
- oral clarithromycin, erythromycin (in pregnancy) or doxycycline is recommended in patients allergic to penicillin

Eron Class II

- NICE recommend: '*Admission may not be necessary if the facilities and expertise are available in the community to give intravenous antibiotics and monitor the person - check local guidelines.*'

Eron Class III-IV

- admit
- NICE recommend: oral/IV co-amoxiclav, oral/IV clindamycin, IV cefuroxime or IV ceftriaxone



Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2019 Cellulitis and erysipelas: antimicrobial prescribing

9 4

Clinical Knowledge Summaries

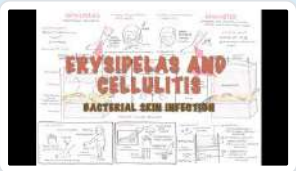
Cellulitis guidelines

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Suggest link

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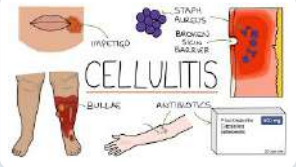
Media



Cellulitis and Erysipelas

Armando Hasudungan - YouTube

1 0



Understanding Cellulitis: Skin and Soft Tissue Infections

Zero To Finals - YouTube

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Report broken media

Score: **19%**

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A 14 month-old Syrian girl is brought to the health centre by her mother. They are living in a Jordanian refugee camp after fleeing violence in their country. She has a widespread maculopapular rash that first affected the face before spreading to the torso and limbs. On examination, she has a cough, rhinorrhoea and conjunctivitis bilaterally. White spotting is seen on her buccal mucosa. The mother reports that her other children have had similar symptoms.

Which is the most appropriate therapy?

- Azithromycin
- Vitamin A
- Vitamin B1 (thiamine)
- Aciclovir
- Reassurance

Submit answer

Reference ranges

Score: **16.7%**

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Which is the most appropriate therapy?

Azithromycin	7%
Vitamin A	29%
Vitamin B1 (thiamine)	3%
Aciclovir	9%
Reassurance	52%

The child has a classical case of measles, showing signs of the three C's (Cough, Coryza & Conjunctivits). Although usually self limiting, measles can have severe consequences. All children in developing countries should receive two dose of vitamin A. This replenishes the low levels of vitamin A caused be measles which can lead on to diarrhoea, corneal perforation and secondary bacterial pneumonia. Vitamin A has been shown to reduce deaths from measles by 50%.

Passive immunity from maternal antibodies protects infants under the age of 1 from measles.

Discuss (11) Improve

Next question

Measles

Measles is now rarely seen in the developed world following the adoption of immunisation programmes. Outbreaks are occasionally seen, particularly when vaccinations rates drop, for example after the MMR controversy of the early 2000s.

Overview

- RNA paramyxovirus

- one of the most infectious known viruses
- spread by aerosol transmission
- infective from prodrome until 4 days after rash starts
- incubation period = 10-14 days

Features

- prodromal phase
  - irritable
  - conjunctivitis
  - fever
- Koplik spots
  - typically develop before the rash
  - white spots ('grain of salt') on the buccal mucosa
- rash
  - starts behind ears then to the whole body
  - discrete maculopapular rash becoming blotchy & confluent
  - desquamation that typically spares the palms and soles may occur after a week
- diarrhoea occurs in around 10% of patients



© Image used on license from DermNet NZ

Koplik spots

Investigations

- IgM antibodies can be detected within a few days of rash onset

Management

- mainly supportive
- admission may be considered in immunosuppressed or pregnant patients
- notifiable disease → inform public health

Complications

- otitis media: the most common complication

- pneumonia: the most common cause of death
- encephalitis: typically occurs 1-2 weeks following the onset of the illness)
- subacute sclerosing panencephalitis: very rare, may present 5-10 years following the illness
- febrile convulsions
- keratoconjunctivitis, corneal ulceration
- diarrhoea
- increased incidence of appendicitis
- myocarditis



© Image used on license from DermNet NZ

The rash typically starts behind the ears and then spreads to the whole body

Management of contacts

- if a child not immunized against measles comes into contact with measles then MMR should be offered (vaccine-induced measles antibody develops more rapidly than that following natural infection)
- this should be given within 72 hours

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 5 6

Subacute sclerosing panencephalitis

Suggest link Report broken link

Media



Measles - What is it?

Let's Learn About Bugs - YouTube 3 0



Measles: Features, Management and Mnemonics

Townsend Teaching - YouTube 2 0



Measles

Osmosis - YouTube 4 1

Report broken media

Score: **19%**

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A 23-year-old white Caucasian left-handed male presents with a 24-hour history of slurred speech. He is a 'frequent flyer' in the Emergency Department with repeated admissions and known is a known user of intravenous drugs and alcohol. His past medical history includes depression and previously treated in adolescence for functional neurological symptoms. He denies having had a drink for the past 2 weeks but used intravenous cocaine yesterday.

On examination, he is obviously unkempt and a lack of prominent veins. There is an old abscess scar over his right femoral crease. His speech is markedly slurred but he remains orientated to time and place. You also note a significant loss of forehead creasing and facial weakness on both sides of his face with normal facial sensation. In addition, his power his 4-/5 in shoulder abduction, shoulder adduction, hip flexion and hip extension, with 5/5 power on all other movements. Reflexes were present in biceps, supinator, patella and ankle, with absent triceps reflex. Both plantar reflexes were downgoing. Sensation to cotton wool, proprioception and pinprick was normal, with the patient reporting no pain.

What is the most likely diagnosis?

- Heavy metal intoxication
- Guillain Barre syndrome
- Botulism
- Chronic inflammatory demyelinating polyneuropathy (CIDP)
- Myasthenia gravis

Submit answer

Reference ranges

Score: **16.7%**

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What is the most likely diagnosis?

Heavy metal intoxication	10%
Guillain Barre syndrome	3%
Botulism	78%
Chronic inflammatory demyelinating polyneuropathy (CIDP)	8%
Myasthenia gravis	2%

The patient has presented with bilateral cranial neuropathies and symmetrical proximal weakness, in the context of intravenous drug use. The descending pattern of weakness is atypical for Guillain-Barre syndrome. The time course of within 8 weeks also rules out CIDP. Myasthenia gravis is a possibility but there is no suggestion of fatigability. The distinction between heavy metal intoxication and botulism is difficult: intravenous drug users, particularly those sourcing from Eastern Europe, who have been known to present with manganese poisoning due to dirty constituents. Clinical features of course depend on the precise metal involved but acute poisoning typically includes encephalopathy, gastrointestinal upset and myalgia in addition to peripheral neuropathy. Significant sensory neuropathy causing neuropathic pain is commonly prominent. In the case of this patient, with bilateral cranial neuropathies and symmetrical proximal weakness, in the context of intravenous drug use, makes botulism most likely. Treatment is with antitoxin, removal of the original source of infection in addition to close monitoring of respiratory function.

Botulism

Clostridium botulinum

- gram positive anaerobic bacillus
- 7 serotypes A-G
- produces botulinum toxin, a neurotoxin which irreversibly blocks the release of acetylcholine
- may result from eating contaminated food (e.g. tinned) or intravenous drug use
- neurotoxin often affects bulbar muscles and autonomic nervous system

Features

- patient usually fully conscious with no sensory disturbance
- flaccid paralysis
- diplopia
- ataxia
- bulbar palsy

Treatment

- botulism antitoxin and supportive care
  - antitoxin is only effective if given early - once toxin has bound its actions cannot be reversed

Textbooks

High-yield textbook

Extended textbook

Score: **19%**

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A 45-year-old male is brought into the emergency department by his partner. He is known to be HIV +ve and his partner reports good compliance with his medications since his diagnosis 4 years ago. A collateral history reveals persistent confusion over the past 3 weeks. His past medical history includes outpatient treatment 2 years ago for lymphogranuloma venereum and type 2 diabetes mellitus. On examination, the patient is not orientated in time or place. He scores 0/10 on the abbreviated mental test. Both heart sounds are present, include a mild early diastolic murmur. Neurological examination is difficult due to poor patient compliance but you note absent reflexes in both lower limbs, with an upgoing plantar on the left and withdrawn plantar on the right. You also note that he has erythematous soles on both feet. Blood tests and blood glucose are awaited. What is the most likely diagnosis?

- ○ Subacute combined degeneration of the cord ✗
- ○ Motor neurone disease ✗
- ○ Neurosyphilis ✗
- ○ Hypoglycaemia and diabetic peripheral neuropathy ✗
- ○ HIV dementia and peripheral neuropathy ✗

Submit answer

Reference ranges

Score: **16.7%**

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Subacute combined degeneration of the cord	7%
Motor neurone disease	1%
Neurosyphilis	72%
Hypoglycaemia and diabetic peripheral neuropathy	2%
HIV dementia and peripheral neuropathy	18%

A number of features suggest neurosyphilis from the history. Firstly, the likely aortic regurgitation on cardiovascular examination should make you suspicious of aortitis, of which syphilitic infection is a classic cause. He has had a range of sexually transmitted diseases. A painless red rash is also a classic sign of syphilis, particularly in palms of the hands or the soles of the feet. He also has a combined upgoing plantar with absent ankle jerks, which narrows the diagnosis to the classic five MRCP favourite of motor neurone disease, tabes dorsalis, subacute combined degeneration of the cord, Friedrich's ataxia and dual pathology of central and peripheral causes. There is little to suggest a genetic disorder presenting in his mid-40s or a vitamin B deficiency to account for SCDC. Neurosyphilis would be the most likely diagnosis.

Discuss (5) Improve

Next question

Syphilis

Syphilis is a sexually transmitted infection caused by the spirochaete *Treponema pallidum*. Infection is characterised by primary, secondary and tertiary stages. The incubation period is between 9-90 days

Primary features

- chancre - painless ulcer at the site of sexual contact
- local non-tender lymphadenopathy
- often not seen in women (the lesion may be on the cervix)

Secondary features - occurs 6-10 weeks after primary infection

- systemic symptoms: fevers, lymphadenopathy
- rash on trunk, palms and soles
- buccal 'snail track' ulcers (30%)
- condylomata lata (painless, warty lesions on the genitalia)





Tertiary features

- gummas (granulomatous lesions of the skin and bones)
- ascending aortic aneurysms
- general paralysis of the insane
- tabes dorsalis
- Argyll-Robertson pupil

Features of congenital syphilis

- blunted upper incisor teeth (Hutchinson's teeth), 'mulberry' molars
- rhagades (linear scars at the angle of the mouth)
- keratitis
- saber shins
- saddle nose
- deafness

AI 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermIS.net 5 5

Picture of condylomata lata

Clinical Knowledge Summaries 8 4

Syphilis guidelines

DermNet NZ 12 5

Syphilis images

Suggest link Report broken link

Media



Syphilis

Osmosis - YouTube 5 0

Report broken media

Score: **19%**

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Back to top

A 22-year-old lady, with lupus and antiphospholipid syndrome, presented to the Rheumatology clinic with sharp chest pain across her right side.

This pain had started 3 days prior to presentation. She had some shortness of breath. She reported that she has a vesicular rash that had started 1 day ago over that area.

She weighs 65KG

Her medications include:

Methotrexate 20mg once weekly
Prednisolone 30mg once a day
Aspirin 75mg once a day
Folic Acid 5mg once a day

She was admitted to the rheumatology ward and underwent a CT pulmonary angiography which showed no pulmonary emboli, but widespread bilateral changes of subsolid nodules and ground-glass opacification.

Her shortness of breath started to get worse and her observations were taken.

Observations:

Temperature	39.1ºC
Blood pressure	107/55mmHg
Heart rate	122beats/min
Oxygen saturation	88% on air

What is the best treatment for her likely diagnosis?

- Treat with dalteparin subcutaneously
- Treat with intravenous amoxicillin and oral clarithromycin
- Treat with intravenous amoxicillin
- Treat with aciclovir intravenous

Treat with aciclovir orally

Submit answer

Reference ranges

Score: **16.7%**

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Observations:

Temperature	39.1ºC
Blood pressure	107/55mmHg
Heart rate	122beats/min
Oxygen saturation	88% on air

What is the best treatment for her likely diagnosis?

Treat with dalteparin subcutaneously	3%
Treat with intravenous amoxicillin and oral clarithromycin	13%
Treat with intravenous amoxicillin	3%
Treat with aciclovir intravenous	73%

Treat with aciclovir orally 8%

The most likely diagnosis for this immunocompromised lady with sharp pain and a vesicular rash is varicella zoster chickenpox. She is systemically unwell with it and has widespread pulmonary changes that are consistent with varicella pneumonitis.

The treatment of choice in an immunocompromised host with varicella zoster is intravenous aciclovir.

Answers 1,2 and 3 would not treat the varicella zoster infection.

https://www.britishinfection.org/files/9114/1617/4223/jeffery08VZV0.pdf

Discuss (5) Improve

Next question

Chickenpox

Chickenpox is caused by primary infection with varicella zoster virus. Shingles is a reactivation of the dormant virus in dorsal root ganglion

Chickenpox is highly infectious

- spread via the respiratory route
- can be caught from someone with shingles
- infectivity = 4 days before rash, until 5 days after the rash first appeared*
- incubation period = 10-21 days

Clinical features (tend to be more severe in older children/adults)

- fever initially
- itchy, rash starting on head/trunk before spreading. Initially macular then papular then vesicular
- systemic upset is usually mild

Management is supportive

- keep cool, trim nails
- calamine lotion
- school exclusion: NICE state the following: *Advise that the most infectious period is 1-2 days before the rash appears, but infectivity continues until all the lesions are dry and have crusted over (usually about 5 days after the onset of the rash).*

- immunocompromised patients and newborns with peripartum exposure should receive varicella zoster immunoglobulin (VZIG). If chickenpox develops then IV aciclovir should be considered

A common complication is secondary bacterial infection of the lesions

- NSAIDs may increase this risk
- whilst this commonly may manifest as a single infected lesion/small area of cellulitis, in a small number of patients invasive group A streptococcal soft tissue infections may occur resulting in necrotizing fasciitis

Rare complications include

- pneumonia
- encephalitis (cerebellar involvement may be seen)
- disseminated haemorrhagic chickenpox
- arthritis, nephritis and pancreatitis may very rarely be seen



© Image used on license from Radiopaedia

Chest x-ray showing miliary opacities secondary to healed varicella pneumonia. Multiple tiny calcific miliary opacities noted throughout both lungs. These are of uniform size and dense suggesting calcification. There is no focal lung parenchymal mass or cavitating lesion seen. The appearances are characteristic for healed varicella pneumonia.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Greenbook 8 6

Varicella

Public Health England 8 6

Chapter 9: managing specific infectious diseases

Suggest link Report broken link

Media



Varicella zoster virus

Osmosis - YouTube 6 0

Report broken media

Score: **19%**

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A 32-year-old lady of Ugandan origin presents to the HIV clinic.

She has been known to the HIV service since coming to the UK 10 years ago. She currently takes tenofovir, emtricitabine, and dolutegravir. She is extremely compliant with her current regime and is currently well.

She is 14 weeks pregnant and this is her first pregnancy. Despite some vomiting earlier in the pregnancy, she is excited and feeling well.

Her blood tests show:

CD4 count	0.84x10^9/L
CD8 count	1.01x10^9/L
Viral load	<20RNA/ml

She has come to discuss the management of her HIV with you.

What is the most appropriate management for this lady?

- Stop this patients current regime and change her onto zidovudine, lamivudine and nevirapine
- Continue the current medications, but at a lower dose so that the medications do not cross the placental barrier and effect the foetus
- Continue current regime and advise patient she will need to deliver by caesarean section to prevent transmission during birth
- Continue current management and advise patient there is currently no reason why she should not have a vaginal delivery
- Stop all HIV medications and advise this patient she will have to deliver by caesarean section

Submit answer

Reference ranges

Score: **16.7%**

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CD8 count	1.01x10^9/L
Viral load	<20RNA/ml

She has come to discuss the management of her HIV with you.

What is the most appropriate management for this lady?

Stop this patients current regime and change her onto zidovudine, lamivudine and nevirapine	5%
Continue the current medications, but at a lower dose so that the medications do not cross the placental barrier and effect the foetus	2%
Continue current regime and advise patient she will need to deliver by caesarean section to prevent transmission during birth	31%
Continue current management and advise patient there is currently no reason why she should not have a vaginal delivery	60%
Stop all HIV medications and advise this patient she will have to deliver by caesarean section	2%

It is recommended that women conceiving on an effective antiretroviral (ART) regimen should continue this even if it contains efavirenz or does not contain zidovudine.

Exceptions are: (1) Protease inhibitor (PI) monotherapy should be intensified to include (depending on tolerability, resistance and prior antiretroviral history) one or more agents that cross the

placenta. (2) The combination of stavudine and didanosine should not be prescribed in pregnancy

Although there is most evidence and experience in pregnancy with zidovudine plus lamivudine, tenofovir plus emtricitabine or abacavir plus lamivudine are acceptable nucleoside backbones. In the absence of specific contraindications, it is recommended that the third agent in ART should be efavirenz or nevirapine (if the CD4 cell count is less than 250 cells/L) or a boosted PI.

No routine dose alterations are recommended for ARVs during pregnancy if used at adult licensed doses.

With a viral load <50 at 36 weeks a vaginal delivery is recommended in the absence of any obstetric complications. Since her viral load has been undetectable for the last 6 years it is reasonable to assume this is likely to continue throughout her pregnancy. She will continue to need monitoring of her viral load throughout her pregnancy, particularly at 36 weeks.

In 2015 there were an estimated 1.5 million people living in Uganda with HIV.

http://www.unaids.org/en/regionscountries/countries/uganda
http://www.bhiva.org/documents/Guidelines/Pregnancy/2012/BHIVA-Pregnancy-guidelines-update-2014.pdf

Discuss (2) Improve

Next question >

HIV and pregnancy

With the increased incidence of HIV infection amongst the heterosexual population there are an increasing number of HIV positive women giving birth in the UK. In London the incidence may be as high as 0.4% of pregnant women. The aim of treating HIV positive women during pregnancy is to minimise harm to both the mother and fetus, and to reduce the chance of vertical transmission.

Guidelines regularly change on this subject and most recent guidelines can be found using the links provided.

Factors which reduce vertical transmission (from 25-30% to 2%)

- maternal antiretroviral therapy
- mode of delivery (caesarean section)
- neonatal antiretroviral therapy
- infant feeding (bottle feeding)

Screening

- NICE guidelines recommend offering HIV screening to all pregnant women

Antiretroviral therapy

- all pregnant women should be offered antiretroviral therapy regardless of whether they were taking it previously

Mode of delivery

- vaginal delivery is recommended if viral load is less than 50 copies/ml at 36 weeks, otherwise caesarian section is recommended
- a zidovudine infusion should be started four hours before beginning the caesarean section

Neonatal antiretroviral therapy

- zidovudine is usually administered orally to the neonate if maternal viral load is <50 copies/ml. Otherwise triple ART should be used. Therapy should be continued for 4-6 weeks.

Infant feeding

- in the UK all women should be advised not to breast feed



Next question

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

3 2

2014 HIV in pregnancy guidelines

BHIVA

6 2

2013 Useful patient orientated leaflet

Patient.info

8 1

HIV and pregnancy

Suggest link

Report broken link

Score: **19%**

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Back to top

An 80-year-old gentleman presented with a 4 month history of feeling generally unwell, being more breathless and tired than usual, feeling feverish and having a dry cough. He had already been treated by the General Practitioner with 2 courses of antibiotics in the community with only temporary improvement. His past medical history includes previous myocardial infarction, permanent pacemaker (PPM) with box-change 8 months ago, hypertension, diabetes, anaemia and chronic kidney disease (stage 2). He is a lifelong smoker.

On examination his heart sounds were normal with no murmurs, he had scattered crackles and his abdomen was soft and non-tender. There was mild leg oedema and a faint purpuric rash on his shins. His vital signs revealed heart rate = 80 beats per minute, blood pressure = 130/70 mmHg, T=37.8oC, SaO2 = 96% on air and respiratory rate = 20 breaths per minute. His chest X-ray did not show and consolidation and his urine was clear.

The following blood tests have been obtained:

Hb	10.5 g/dl
MCV	95 fl
Platelets	160 * 10^9/l
WBC	13.4 * 10^9/l

Na^+	132 mmol/l
K^+	4.9 mmol/l
Urea	12 mmol/l
Creatinine	150 µmol/l
CRP	100 mg/l

Blood cultures grow coagulase negative staphylococci and you notice that during his previous admission in the hospital he also had positive blood cultures for coagulase-negative staphylococci.

What is the best next investigation?

- Repeat blood cultures
- Computed tomography thorax / abdomen / pelvis
- Biopsy of the rash

- Vasculitic screen
- Urgent transoesophageal echocardiogram

Submit answer

Reference ranges

Score: **16.7%**

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Blood cultures grow coagulase negative staphylococci and you notice that during his previous admission in the hospital he also had positive blood cultures for coagulase-negative staphylococci.

What is the best next investigation?

Repeat blood cultures	8%
Computed tomography thorax / abdomen / pelvis	10%
Biopsy of the rash	5%

Vasculitic screen	6%
Urgent transoesophageal echocardiogram	71%

This question aims to raise awareness of the significance of blood cultures positive for coagulase-negative staphylococci.

Coagulase-negative staphylococci are often considered skin contaminant or apathogenic organisms. However, in the presence of prosthetic devices such as PPM, prosthetic heart valves, central venous lines and orthopaedic prostheses they are the commonest cause of infection causing device-related infection. This is because of their ability to establish biofilms on artificial layers. *Staphylococcus epidermidis* device-related infections: pathogenesis and clinical management, JPP 2008, 60:1551-1571

This patient presented with symptoms and sings suggestive of a chronic infection and had blood cultures positive for coagulase-negative staphylococci. The presence of the PPM and the purpuric rash indicate that the source of sepsis is possibly related to the medical device. The box-change also increases the risk of infection.

Repeating the blood cultures is a good idea but it does not help identify the source of sepsis. CT thorax / abdomen / pelvis is an investigation that can identify most sources of sepsis especially if there is a clinical suspicion of lung or intra-abdominal abscess. However, in this case, it will not be particularly useful to verify the source of sepsis. Vasculitis is less likely in that scenario and so biopsy of the rash or vasculitic screen should not be the next investigation.

Transoesophageal echocardiogram is the best investigation to rule out pacemaker-wire related infections. It requires a lot of expertise and it might not be readily available but even so it is the next best investigation in this scenario. Please see the following article for further information. Update on Cardiovascular Implantable Electronic Device Infections and Their Management: A Scientific Statement From the American Heart Association, Circulation. 2010; 121: 458-477

Discuss (3) Improve

Next question ›

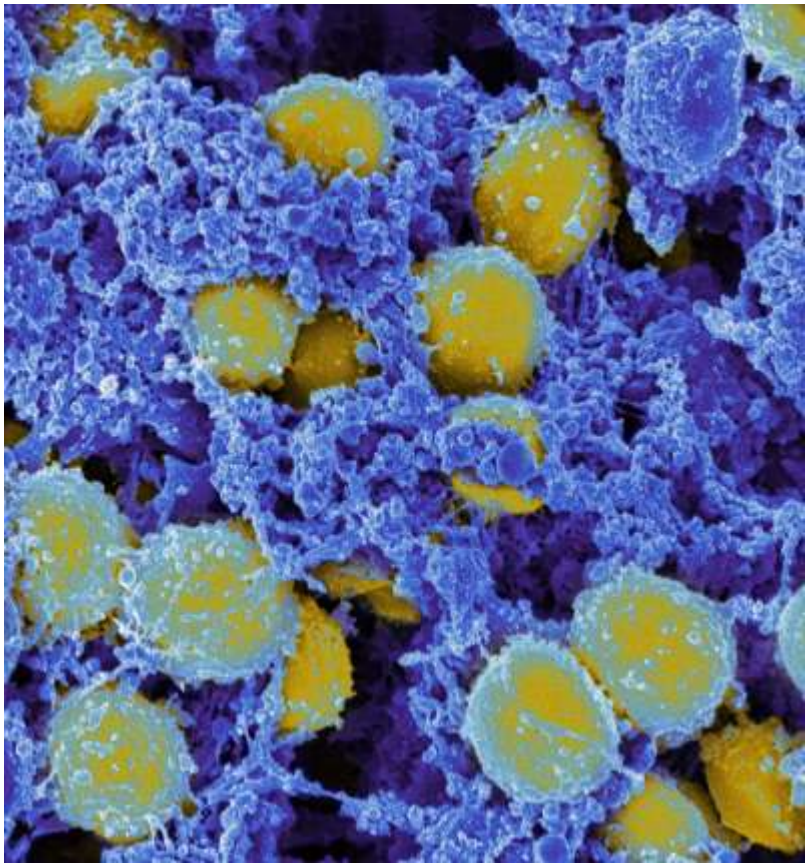
Staphylococci

Staphylococci are a common type of bacteria that are often found normal commensal organisms but may also cause invasive disease. Some basic facts include:

- Gram-positive cocci
- facultative anaerobes
- produce catalase

The two main types of Staphylococci you need to know about are *Staphylococcus aureus* and *Staphylococcus epidermidis*.

Staphylococcus aureus	***Staphylococcus epidermidis***
• Coagulase-positive • Causes skin infections (e.g. cellulitis), abscesses, osteomyelitis, toxic shock syndrome	• Coagulase-negative • Cause of central line infections and infective endocarditis



Scanning electromicrograph of Staphylococcus aureus bacteria.
Credit: NIAID

Next question

Textbooks

High-yield textbook

Extended textbook

Media



Staphylococcus species (Characteristics, Clinical complications, Differences)

ATP - YouTube 7 0

Report broken media

Score: **19%**

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A 19-year-old from Cameroon has moved to the UK to study nursing. She arrived three months ago. She has been having diarrhoea for a month and noticed a fleeting erythematous rash on her torso. Her GP orders a set of bloods, which reveal a dramatic eosinophilia. She has not lost weight and, if anything, seems concerned that she is becoming overweight since moving to the UK. She has been well previously with no allergies or medication. There is no significant family history but she tells you that her brother had an 'eye worm' last year.

A stool is sent for ova cysts and parasites and microscopy and culture. Multiple *Strongyloides stercoralis* larvae can be seen on charcoal culture. She is commenced on a seven-day course of Ivermectin.

Four days later she is brought into the Emergency Department, with a GCS of 6.

What is the diagnosis?

- Disseminated *Strongyloides* infection ×
- Co-infection with Onchocerciasis ×
- Co-infection with Loa ×
- Ivermectin related hypoglycaemia ×
- Insulin abuse ×

Submit answer

Reference ranges

Score: **16.7%**

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A stool is sent for ova cysts and parasites and microscopy and culture. Multiple *Strongyloides stercoralis* larvae can be seen on charcoal culture. She is commenced on a seven-day course of Ivermectin.

Four days later she is brought into the Emergency Department, with a GCS of 6.

What is the diagnosis?

Disseminated *Strongyloides* infection	30%
Co-infection with Onchocerciasis	16%
Co-infection with Loa	23%
Ivermectin related hypoglycaemia	29%
Insulin abuse	2%

The give away in this history is the family history of 'eye worm'. This suggests that she lives in an area of high Loa Loa endemicity.

Discuss (11) | Improve

Next question

Loiasis

Loiasis is a filarial infection caused by Loa Loa. It is transmitted by the Chrysops deerfly and tends to occur in rainforest regions of Western and Central Africa.

Clinical features

- pruritus
- urticaria
- Calabar swellings: transient, non-erythematous, hot swelling of soft-tissue around joints
- 'eye worm' - the dramatic presentation of subconjunctival migration of the adult worm.

It has fewer pathological features than other the microfilarial infections Onchocerciasis and Lymphatic Filariasis. However high loa loa microfilaraemia is associated with encephalopathy following treatment with either Ivermectin or DEC. This occurs due to the death of vast numbers of blood microfilaria. Both of these drugs are contraindicated if loa loa microfilaraemia exceeds 2500 mf/ml.

This has significant public health implications as Ivermectin is currently the drug of choice for control of both Onchocerciasis and Lymphatic Filariasis in Africa.



Adult Loa loa parasite. Loa loa is the filarial nematode (roundworm) species that causes loa loa filariasis. It is commonly known as the 'eye worm.' Its geographic distribution includes Africa and India. Credit: NIAID

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **19%**

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Back to top

A 24-year-old Caucasian girl returned from a two week scuba diving holiday in Honduras four days ago. She had not taken malaria prophylaxis. She had been drinking alcohol moderately during her trip. She had unprotected sex during her holiday with her diving instructor.

Yesterday she had felt hot and cold with pain all over her body and a headache. Today the pain is worst behind her eyes. It is a constant pain and there is no photophobia or neck stiffness. She has an erythematous rash over her trunk and back with patches of white skin surrounded by erythema. There is no lymphadenopathy

She is observed in hospital for 48 hours and tests for HIV (including PCR and antibody), malaria and blood cultures were negative. Further tests remain unreported.

She recovers and is feeling much better. Prior to discharge, she asks if she is safe to go back to Honduras next year.

What should you advise?

- She should not go back to any Dengue endemic area as there is a risk of Dengue haemorrhagic fever (with a 20% mortality) with a second exposure to Dengue virus
- She can go back to Honduras, although there is a small risk of Dengue haemorrhagic fever with a second exposure, the risk is small enough not to be overly worried. She should take precautions not to be bitten by mosquitos during the day
- She has had Dengue fever so will be immune to Dengue fever on subsequent exposure
- She has had flu and therefore she can go back to Honduras next year
- Staying in the UK from now on would be the most sensible given that she is at high risk of malaria, HIV and Dengue fever from her behavioral tendencies

Submit answer

Reference ranges

Score: **16.7%**

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She is observed in hospital for 48 hours and tests for HIV (including PCR and antibody), malaria and blood cultures were negative. Further tests remain unreported.

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What should you advise?

She should not go back to any Dengue endemic area as there is a risk of Dengue haemorrhagic fever (with a 20% mortality) with a second exposure to Dengue virus	31%
She can go back to Honduras, although there is a small risk of Dengue haemorrhagic fever with a second exposure, the risk is small enough not to be overly worried. She should take precautions not to be bitten by mosquitos during the day	61%
She has had Dengue fever so will be immune to Dengue fever on subsequent exposure	4%
She has had flu and therefore she can go back to Honduras next year	1%
Staying in the UK from now on would be the most sensible given that she is at high risk of malaria, HIV and Dengue fever from her behavioral tendencies	4%

There are four serovars of Dengue (1-4) and therefore infection with one serovar will not confer immunity to the other serovars. There is a theoretical risk that consequent infection with serovar 2 following infection with serovar 1 will increase your risk of progression to Dengue Haemorrhagic Fever. However, the risk is far smaller than other risks posed by travelling (e.g road traffic accident, acquiring HIV from unprotected sex etc.) Therefore universal travel advice (including avoidance of day biting mosquitos) is appropriate.

Discuss (8) | Improve

Next question >

Dengue fever

Dengue fever is a viral infection that can progress to viral haemorrhagic fever (other examples include yellow fever, Lassa fever, Ebola).

Aetiology

- dengue virus is a RNA virus of the genus Flavivirus
- transmitted by the *Aedes aegypti* mosquito
- incubation period of 7 days

Clinical features

Patients with dengue fever can be classified as follows:

- dengue fever:
  - without warning signs
  - with warning signs
- severe dengue (dengue haemorrhagic fever)

Dengue fever

- fever
- headache (often retro-orbital)
- myalgia, bone pain and arthralgia ('break-bone fever')
- pleuritic pain
- facial flushing (dengue)
- maculopapular rash
- haemorrhagic manifestations e.g. positive tourniquet test, petechiae, purpura/ecchymosis, epistaxis
- 'warning signs' include:
  - abdominal pain
  - hepatomegaly
  - persistent vomiting
  - clinical fluid accumulation (ascites, pleural effusion)

Severe dengue (dengue haemorrhagic fever)

- this is a form of disseminated intravascular coagulation (DIC) resulting in:
  - thrombocytopenia
  - spontaneous bleeding

- around 20-30% of these patients go on to develop dengue shock syndrome (DSS)

Investigations

- typically blood results
  - leukopenia, thrombocytopenia, raised aminotransferases
- diagnostic tests
  - serology
  - nucleic acid amplification tests for viral RNA
  - NS1 antigen test

Treatment

- entirely symptomatic e.g. fluid resuscitation, blood transfusion etc
- no antivirals are currently available



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 8 14

2011 Dengue fever review

Postgraduate Medical Journal 6 9

Review of dengue fever

Suggest link Report broken link

Score: **19%**

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You are called to see a 50-year-old Indian gentleman who has presented to the Emergency Department (ED) from Heathrow airport. He gives a 3 week history of a brown productive cough associated with fevers and drenching nights sweats. He denies any weight loss or haemoptysis, however his wife is concerned that his sputum has been very brown/red in colour since his illness started. On further questioning you find that the patient is a resident of a rural community in north east India and is visiting family who live in London. He is a non-smoker and had been in very good health apart from a bout of diarrhoea several weeks ago.

On examination of the patient he appears relatively well, his heart rate is 95 beats per minute and respiratory rate is 20 breaths per minute. You note that he has a productive cough, producing dark brown sputum which you send for routine microscopy sensitivity and culture (MC&S) and acid fast bacilli (AFB) and tuberculosis PCR. On auscultation of his chest you hear course crepitations at the right base with some bronchial breathing. The remainder of his physical examination is unremarkable.

You perform a number of investigations described below

Hb	110 g/l	Na^+	138 mmol/l
Platelets	500 * 10^9/l	K^+	4.5 mmol/l
WBC	16.0 * 10^9/l	Urea	6.0 mmol/l
Neuts	10.0 * 10^9/l	Creatinine	99 µmol/l
Lymphs	2.0 * 10^9/l	CRP	55 mg/l
Eosin	2.0 * 10^9/l		
Sputum MC&S	Negative		
Sputum AFB	Negative		
Chest X-Ray	Right lower zone consolidation		

What is the likely diagnosis?

- Tuberculosis
- Streptococcal pneumonia
- Paragonimiasis
- Malignancy

Pulmonary Haemorrhage

Submit answer

Reference ranges

Score: **16.7%**

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Lymphs	2.0 * 10^9/l	CRP	55 mg/l
Eosin	2.0 * 10^9/l		
Sputum MC&S	Negative		
Sputum AFB	Negative		
Chest X-Ray	Right lower zone consolidation		

What is the likely diagnosis?

Tuberculosis	8%
Streptococcal pneumonia	14%
Paragonimiasis	75%
Malignancy	1%

Pulmonary Haemorrhage 2%

The diagnosis is paragonimiasis or lung fluke infection.

Background

Paragonimiasis is a common tropical infection, the presentation of which can often be misinterpreted as tuberculosis. Patients classically present with a productive cough of brown / red sputum (which can often be mistaken for haemoptysis). Patients may also exhibit constitutional symptoms, however, these are often less severe than in tuberculosis or malignancy. Furthermore, paragonimiasis is often associated with an eosinophilia.

Although more commonly associated with East Asia, paragonimiasis is also prevalent in North East India, Africa and parts of the Americas.

Treatment of paragonimiasis is with praziquantel.

Discuss (7) Improve

Next question

Helminths

Nematodes (roundworms)

Worm	Notes	Treatment
Strongyloides stercoralis	Larvae are present in soil and gain access to the body by penetrating the skin Features include diarrhoea, abdominal pain, papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks, larva currens: pruritic, linear, urticarial rash, if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered	Ivermectin and -bendazoles are used
Enterobius vermicularis (pinworm)	Threadworm infestation is asymptomatic in around 90% of cases, possible features include perianal itching, particularly at night; girls may have vulval symptoms	-bendazoles

Worm	Notes	Treatment
	Diagnosis may be made by the applying sticky plastic tape to the perianal area and sending it to the laboratory for microscopy to see the eggs	
Ancylostoma duodenale, *Necator americanus* (hookworms)	Larvae penetrate skin of feet; gastrointestinal infection → anaemia Thin-shelled ova	-bendazoles
Loa loa	Transmission by deer fly and mango fly Causes red itchy swellings below the skin called 'Calabar swellings', may be observed when crossing conjunctivae	Diethylcarbamazine
Trichinella spiralis	Typically develops after eating raw pork Features include fever, periorbital oedema and myositis (larvae encyst in muscle)	-bendazoles
Onchocerca volvulus	Causes 'river blindness'. Spread by female blackflies Features include blindness, hyperpigmented skin and possible allergic reaction to microfilaria	Ivermectin r**IVER**blindness = **IVER**mectin
Wuchereria bancrofti	Transmission by female mosquito Causes blockage of lymphatics → elephantiasis	Diethylcarbamazine
Toxocara canis (dog roundworm)	Transmitted through ingestion of infective eggs. Features include visceral larva migrans and retinal granulomas VISCious dogs → blindness	Diethylcarbamazine
Ascaris lumbricoides (giant roundworm)	Eggs are visible in faeces May cause intestinal obstruction and occasional migrate to lung (Loffler's syndrome)	-bendazoles

Cestodes (tapeworms)

Worm	Notes	Treatment
Echinococcus granulosus	Transmission through ingestion of eggs in dog faeces. Definite host is dog, which ingests hydatid cysts from sheep, who act as an intermediate host. Often seen in farmers. Features include liver cysts and anaphylaxis if cyst ruptures (e.g. during surgical removal)	- bendazoles
Taenia solium	Often transmitted after eating undercooked pork. Causes cysticercosis and neurocysticercosis, mass lesions in the brain 'swiss cheese appearance'	- bendazoles

Trematodes (flukes)

Worm	Notes	Treatment
Schistosoma haematobium	Hosted by snails, which release cercariae that penetrate skin. Causes 'swimmer's itch' - frequency, haematuria. Risk factor for squamous cell bladder cancer	Praziquantel
Paragonimus westermani	Caused by undercooked crabmeat, results in secondary bacterial infection of lungs	Praziquantel
Clonorchis sinensis	Caused by undercooked fish Features include biliary tract inflammation. Known risk factor for cholangiocarcinoma	Praziquantel
Fasciola hepatica (the liver fluke)	May cause biliary obstruction	Triclabendazole

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

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Examination reveals a comfortable patient with evidence of a right lower zone pneumonia. The is confirmed on chest radiograph. You send a number of investigations including sputum for routine culture and acid fast bacilli. The results of which are shown below:

Hb	120 g/l	Na^+	139 mmol/l
Platelets	350 * 10^9/l	K^+	3.9 mmol/l
WBC	13.0 * 10^9/l	Urea	8.0 mmol/l
Neuts	8.0 * 10^9/l	Creatinine	110 µmol/l
Lymphs	1.5 * 10^9/l	CRP	35 mg/l
Eosin	1.8 * 10^9/l		
Sputum MC&S	Negative		
Sputum TB PCR	Negative		

What is the most appropriate treatment for this patient?

- Rifater and ethambutol
- Amoxicillin
- Praziquantel
- Augmentin and clarithromycin
- Fluconazole

Submit answer

Reference ranges

Score: **16.7%**

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Lymphs	1.5 * 10^9/l	CRP	35 mg/l
Eosin	1.8 * 10^9/l		
Sputum MC&S	Negative		
Sputum TB PCR	Negative		

What is the most appropriate treatment for this patient?

- Rifater and ethambutol — 8%
- Amoxicillin — 7%
- Praziquantel — 52%
- Augmentin and clarithromycin — 22%
- Fluconazole — 11%

In this case the diagnosis is paragonimiasis, or lung fluke infection. Paragonimiasis is a common tropic al disease, which often presents in a similar fashion to tuberculosis. The patient will have a productive cough of classically brown or red sputum (which may be misinterpreted as

haemoptysis). They often will also present with fever and night sweats as well as rashes or urticaria. Constitutional symptoms will often not be as severe as in tuberculosis and the patient may also have an eosinophilia.

Treatment of paragonimiasis is with praziquantel

Discuss (6) | Improve

Next question >

Helminths

Nematodes (roundworms)

Worm	Notes	Treatment
Strongyloides stercoralis	Larvae are present in soil and gain access to the body by penetrating the skin Features include diarrhoea, abdominal pain, papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks, larva currens: pruritic, linear, urticarial rash, if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered	Ivermectin and -bendazoles are used
Enterobius vermicularis (pinworm)	Threadworm infestation is asymptomatic in around 90% of cases, possible features include perianal itching, particularly at night; girls may have vulval symptoms Diagnosis may be made by the applying sticky plastic tape to the perianal area and sending it to the laboratory for microscopy to see the eggs	-bendazoles
Ancylostoma duodenale, *Necator americanus* (hookworms)	Larvae penetrate skin of feet; gastrointestinal infection → anaemia Thin-shelled ova	-bendazoles
Loa loa	Transmission by deer fly and mango fly	Diethylcarbamazine

Worm	Notes	Treatment
	Causes red itchy swellings below the skin called 'Calabar swellings', may be observed when crossing conjunctivae	
Trichinella spiralis	Typically develops after eating raw pork Features include fever, periorbital oedema and myositis (larvae encyst in muscle)	-bendazoles
Onchocerca volvulus	Causes 'river blindness'. Spread by female blackflies Features include blindness, hyperpigmented skin and possible allergic reaction to microfilaria	Ivermectin r**IVER**blindness = **IVER**mectin
Wuchereria bancrofti	Transmission by female mosquito Causes blockage of lymphatics → elephantiasis	Diethylcarbamazine
Toxocara canis (dog roundworm)	Transmitted through ingestion of infective eggs. Features include visceral larva migrans and retinal granulomas VISCious dogs → blindness	Diethylcarbamazine
Ascaris lumbricoides (giant roundworm)	Eggs are visible in faeces May cause intestinal obstruction and occasional migrate to lung (Loffler's syndrome)	-bendazoles

Cestodes (tapeworms)

Worm	Notes	Treatment
Echinococcus granulosus	Transmission through ingestion of eggs in dog faeces. Definite host is dog, which ingests hydatid cysts from sheep, who act as an intermediate host. Often seen in farmers. Features include liver cysts and anaphylaxis if cyst ruptures (e.g. during surgical removal)	-bendazoles

Worm	Notes	Treatment
Taenia solium	Often transmitted after eating undercooked pork. Causes cysticercosis and neurocysticercosis, mass lesions in the brain 'swiss cheese appearance'	-bendazoles

Trematodes (flukes)

Worm	Notes	Treatment
Schistosoma haematobium	Hosted by snails, which release cercariae that penetrate skin. Causes 'swimmer's itch' - frequency, haematuria. Risk factor for squamous cell bladder cancer	Praziquantel
Paragonimus westermani	Caused by undercooked crabmeat, results in secondary bacterial infection of lungs	Praziquantel
Clonorchis sinensis	Caused by undercooked fish Features include biliary tract inflammation. Known risk factor for cholangiocarcinoma	Praziquantel
Fasciola hepatica (the liver fluke)	May cause biliary obstruction	Triclabendazole

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **19%**

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Back to top

As the medical registrar on-call you are called to see a 25-year-old male in the Emergency Department (ED). The young male presented to the ED with a fever and productive cough of green sputum. When you arrive he looks unwell and although alert is a difficult historian.

Concerned, you immediately assess him from head to toe:

Airway	Airway patient, able to speak in sentences
Breathing	Sats 90% on room air, Resp Rate 25 / min, course creps bilaterally on auscultation
Circulation	Heart rate 110 beats per minute, blood pressure 90/55 mm/Hg
Disability	Glasgow coma sclae 15/15, capillary blood glucose 5.5
Exposure	Multiple boils noted distributed widely across the patients body

You inquire about the history of the boils identified on examination. The patient is unsure how long he has had them for, however, mentions that two of his family members recently were prescribed antibiotics by their family GP for similar lesions.

You start the patient on high flow oxygen, gain IV access taking blood cultures and give a fluid bolus. An urgent portable chest X-ray is requested which appears to show bilateral consolidation with multiple cavitating lesions.

What is the most likely diagnosis

- Streptococcal infection
- PVL-producing staphylococcal infection
- Tuberculosis
- Fungal chest infection
- Klebsiella infection

Submit answer

Reference ranges

Score: **16.7%**

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You start the patient on high flow oxygen, gain IV access taking blood cultures and give a fluid bolus. An urgent portable chest X-ray is requested which appears to show bilateral consolidation with multiple cavitating lesions.

What is the most likely diagnosis

Streptococcal infection	7%
PVL-producing staphylococcal infection	74%
Tuberculosis	5%
Fungal chest infection	3%
Klebsiella infection	11%

Panton-Valentine Leukocidin is a pore-forming toxin which is produced by staphylococcus aureus. It has an affinity for white blood cells and the endothelium. Clinically PVL will often present with a necrotising pneumonia, characterised by severe bilateral pneumonia with cavitations on X-ray. Often patients presenting will have a history of boils or necrotic skin lesions. As the staphylococcus is easily transmitted there may be a recent family history of similar infections / boils.

Although all of the answers in this stem could be associated with cavitating pneumonia, the

severity of the clinical presentation and history of boils in the family points towards a PVL-pneumonia.

Discuss (2) | Improve

Next question >

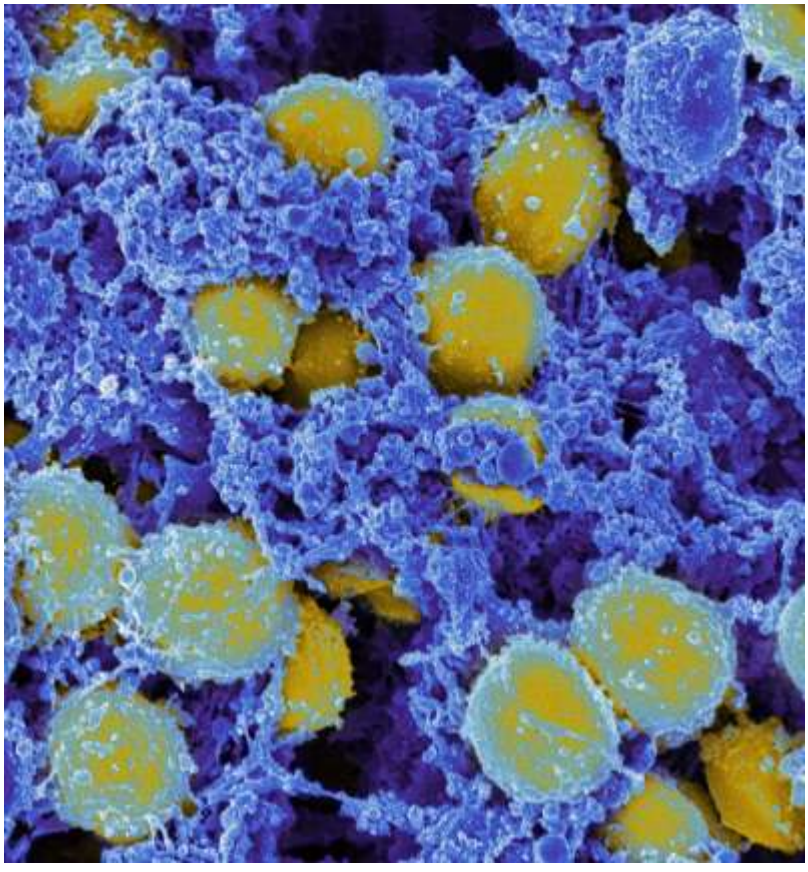
Staphylococci

Staphylococci are a common type of bacteria that are often found normal commensal organisms but may also cause invasive disease. Some basic facts include:

- Gram-positive cocci
- facultative anaerobes
- produce catalase

The two main types of Staphylococci you need to know about are *Staphylococcus aureus* and *Staphylococcus epidermidis*.

Staphylococcus aureus	***Staphylococcus epidermidis***
• Coagulase-positive • Causes skin infections (e.g. cellulitis), abscesses, osteomyelitis, toxic shock syndrome	• Coagulase-negative • Cause of central line infections and infective endocarditis



Scanning electromicrograph of Staphylococcus aureus bacteria.
Credit: NIAID

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media



Staphylococcus species (Characteristics, Clinical complications, Differences)

ATP - YouTube

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Report broken media

Score: **19%**

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A 36-year-old male from India presents with worsening headache and vomiting. He is known to have HIV but has been lost to follow up over the last 5 years. His CD4 count from his last appointment was 400.

On examination he has no localising signs however fundoscopy revealed papiloedema.

Investigations:

haemoglobin	115 g/L (115-165)
white cell count	9.4 Ã— 109/L (4.0-11.0)
platelet count	220 Ã— 109/L (150-400)
erythrocyte sedimentation rate	50 mm/1st h (<30)
serum urea	7.0 mmol/L (2.5-7.0)
serum creatinine	105 mol/L (60-110)
serum alanine aminotransferase	17 U/L (5-35)
serum aspartate aminotransferase	26 U/L (1-31)
Cryptococcal antigen test	Negative
CD4 Count	65 cells/mm3
CT Head	5cm ring enhancing lesion in right temporal lesion with surrounding oedema

What is most appropriate management?

- Amphotericin, Fluconazole and prednisolone
- Rifampicin, Isoniazid, Pyrazinamide, Ethambutol and prednisolone
- Trimethorpim-Sulphamethoxazole, Rifampicin, Isoniazid, Pyrazinamide, Ethambutol and prednisolone
- Start prednisolone but defer treatment until lumbar puncture performed
- Pyrimethamine, Sulfadiazine, Rifampicin, Isoniazid, Pyrazinamide, Ethambutol, and Prednisolone

Submit answer

Reference ranges

Score: **16.7%**

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What is most appropriate management?

Amphotericin, Fluconazole and prednisolone	8%
Rifampicin, Isoniazid, Pyrazinamide, Ethambutol and prednisolone	11%
Trimethorpim-Sulphamethoxazole, Rifampicin, Isoniazid, Pyrazinamide, Ethambutol and prednisolone	13%
Start prednisolone but defer treatment until lumbar puncture performed	14%
Pyrimethamine, Sulfadiazine, Rifampicin, Isoniazid, Pyrazinamide, Ethambutol, and Prednisolone	53%

The question represents a real diagnostic conundrum. You should automatically think of compliance issues given that the patients has defaulted from treatment and is therefore unlikely to have been controlling his HIV. The CD4 count of 400 is therefore not going to be representative of his current immune system.

This leads us to the differential diagnosis of a intra-cerebral ring enhancing lesion in a severely immunocompromised host. This includes tuberculosis, lymphoma, toxoplasmosis and cryptococcus amongst others.

Amphotericin and fluconazole is used in the initial 2 week phase of treatment for cryptococcal meningitis. However we are told that the cryptococcal antigen test is negative. This test has a relatively high sensitivity and specificity is therefore reassuring.

We are also told that the patient has papilloedema and a 5cm lesion with oedema, as a result attempting a lumbar puncture at this early stage is fraught with risk. Although vital to diagnosis it would be prudent to reduce the intra-cerebral swelling first by which time the patient may have seriously deteriorated, therefore empirical treatment becomes a priority.

Diagnosis of CNS tuberculosis is notoriously difficult and without access to CSF it is likely that empirical treatment will be required in the interim. When CSF is available culture, staining and PCR can be used to help detect the organism. Toxoplasmosis is treated with pyrimethamine and sulfadiazine as a first line. Again, empirical treatment may be required before access to CSF is available. As a result the correct answer is treatment of TB and Toxoplasmosis with steroids to reduce oedema.

👍 👎 💬 Discuss (9) | Improve

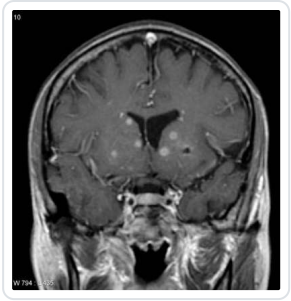
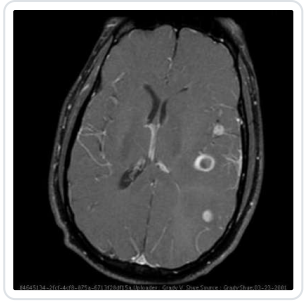
Next question ›

HIV: neurocomplications ★

Focal neurological lesions

Toxoplasmosis

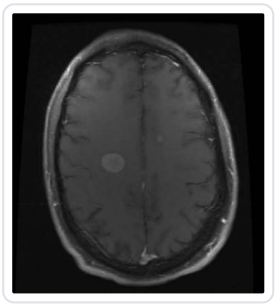
- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination
- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link Report broken link

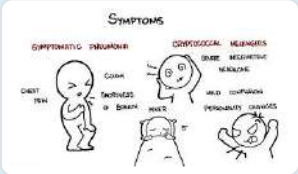
Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

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Cryptococcus neoformans infection

Armando Hasudungan - YouTube

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Report broken media

Score: **19%**

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Back to top

A 36-year-old Somali male presents on the medical take with a severe generalised headache associated with nausea and vomiting. He has been living in the UK for the last 10 years. He was diagnosed with HIV 5 years ago and has been well maintained on therapy with an undetectable viral load and a CD4 count of 500 cells/mm3.

He has a long history of headaches since the age of 10 which are normally controlled with simple analgesia. Over the last 2 days he has been suffering with a particularly bad attack which culminated in him becoming aggressive and vomiting profusely. He was noted to have a tonic-clonic seizure whilst in accident and emergency which resolved with diazepam.

Clinical examination reveals a pulse of 78 beats per minute, a blood pressure of 130/90, oxygen saturations of 98% on air and a temperature of 36.8 degrees. There was no focal neurological defects.

A CT scan subsequently showed cystic and calcified lesions within the brain and mild hydrocephalus.

What is the most likely diagnosis?

- Tuberculous meningitis
- Cryptococcal meningitis
- Lymphoma
- Neurocysticercosis
- Human african trypanosomiasis

Submit answer

Reference ranges

Score: **16.7%**

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A CT scan subsequently showed cystic and calcified lesions within the brain and mild hydrocephalus.

What is the most likely diagnosis?

Tuberculous meningitis	4%
Cryptococcal meningitis	10%
Lymphoma	4%
Neurocysticercosis	80%
Human african trypanosomiasis	2%

Cysticercosis is caused by the larval stage of the tapeworm Taenia solium. Clinical syndromes that can arise include neurocysticercosis (NCC) and extraneural cysticercosis. In endemic areas NCC is an important cause of adult-onset seizures.

Cysticercosis is transmitted by ingestion of T. solium eggs shed in the stool of a human tapeworm carrier. Following ingestion, embryos (oncospheres) hatch in the small intestine, invade the bowel wall and disseminate hematogenously to the brain, muscle, liver and other tissues. Cysts located in the brain result in neurocysticercosis and can lead to hydrocephalus if they obstruct the outflow of CSF from the ventricles. The condition can be chronic and develop over a protracted period of time.

This question relies primarily on knowledge of the relevant condition and your ability to think beyond the fact that the patient has HIV. While the other options are all plausible you are given clues throughout the question on which you can discount each one until you are left with the correct answer.

The first hint is that his HIV was diagnosed early and is very well controlled. This makes an opportunistic infection incredibly unlikely therefore discounting crypotcocosis and TB meningitis. There are two forms of human african trypanosomiasis both transmitted by the tsetse fly, T.gambiense a chronic condition contracted in west africa and T.rhodiesiense that is acutely progressive and contracted in east africa. Given that he has been in the UK for 5 years we can discount T.rhodesiense and the finding of calcified lesions on the CT would be most unusual for T.gambiense.

This leaves us with lymphoma and neurocistercicosis, lymphoma will generally present with progressive focal neurology over a period of time and is unlikely to show calcified lesions in the brain.

Discuss (5) | Improve

Next question >

Helminths ★

Nematodes (roundworms)

Worm	Notes	Treatment
Strongyloides stercoralis	Larvae are present in soil and gain access to the body by penetrating the skin Features include diarrhoea, abdominal pain, papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks, larva currens: pruritic, linear, urticarial rash, if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered	Ivermectin and -bendazoles are used
Enterobius vermicularis (pinworm)	Threadworm infestation is asymptomatic in around 90% of cases, possible features include perianal itching, particularly at night; girls may have vulval symptoms	-bendazoles

Worm	Notes	Treatment
	Diagnosis may be made by the applying sticky plastic tape to the perianal area and sending it to the laboratory for microscopy to see the eggs	
Ancylostoma duodenale, *Necator americanus* (hookworms)	Larvae penetrate skin of feet; gastrointestinal infection → anaemia Thin-shelled ova	-bendazoles
Loa loa	Transmission by deer fly and mango fly Causes red itchy swellings below the skin called 'Calabar swellings', may be observed when crossing conjunctivae	Diethylcarbamazine
Trichinella spiralis	Typically develops after eating raw pork Features include fever, periorbital oedema and myositis (larvae encyst in muscle)	-bendazoles
Onchocerca volvulus	Causes 'river blindness'. Spread by female blackflies Features include blindness, hyperpigmented skin and possible allergic reaction to microfilaria	Ivermectin r**IVER**blindness = **IVER**mectin
Wuchereria bancrofti	Transmission by female mosquito Causes blockage of lymphatics → elephantiasis	Diethylcarbamazine
Toxocara canis (dog roundworm)	Transmitted through ingestion of infective eggs. Features include visceral larva migrans and retinal granulomas VISCious dogs → blindness	Diethylcarbamazine
Ascaris lumbricoides (giant roundworm)	Eggs are visible in faeces May cause intestinal obstruction and occasional migrate to lung (Loffler's syndrome)	-bendazoles

Cestodes (tapeworms)

Worm	Notes	Treatment
Echinococcus granulosus	Transmission through ingestion of eggs in dog faeces. Definite host is dog, which ingests hydatid cysts from sheep, who act as an intermediate host. Often seen in farmers. Features include liver cysts and anaphylaxis if cyst ruptures (e.g. during surgical removal)	- bendazoles
Taenia solium	Often transmitted after eating undercooked pork. Causes cysticercosis and neurocysticercosis, mass lesions in the brain 'swiss cheese appearance'	- bendazoles

Trematodes (flukes)

Worm	Notes	Treatment
Schistosoma haematobium	Hosted by snails, which release cercariae that penetrate skin. Causes 'swimmer's itch' - frequency, haematuria. Risk factor for squamous cell bladder cancer	Praziquantel
Paragonimus westermani	Caused by undercooked crabmeat, results in secondary bacterial infection of lungs	Praziquantel
Clonorchis sinensis	Caused by undercooked fish Features include biliary tract inflammation. Known risk factor for cholangiocarcinoma	Praziquantel
Fasciola hepatica (the liver fluke)	May cause biliary obstruction	Triclabendazole



Next question

Textbooks

High-yield textbook

Extended textbook

Score: **19%**

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A 69-year-old man from Pakistan presents to his general practitioner with a three month history of cough associated with episodes of haemoptysis and has had some episodes of shortness of breath. His only past medical history of note is asthma for which he takes salbutamol inhaler as required and 2 puffs of beclomethasone inhaler twice daily. He has a 25 pack-year history and he is teetotal. The patient's GP performs blood tests and refers the patient to the rapid access chest clinic.

Blood test results come back as all within normal reference ranges, however, chest x-ray shows a solid lesion at the apex of the right lung, that is associated with a rim of air.

Of the following options, which is the most likely diagnosis?

- Tuberculosis
- Mesothelioma
- Aspergilloma
- Squamous cell carcinoma
- Small cell lung carcinoma

Submit answer

Reference ranges

Score: **16.7%**

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Blood test results come back as all within normal reference ranges, however, chest x-ray shows a solid lesion at the apex of the right lung, that is associated with a rim of air.

Of the following options, which is the most likely diagnosis?

Tuberculosis	19%
Mesothelioma	1%
Aspergilloma	76%
Squamous cell carcinoma	3%
Small cell lung carcinoma	2%

The most likely diagnosis in this case is an aspergilloma - a fungal ball located in a previous cavity in the lung, as seen by the solid mass coupled with the rim of air and the haemoptysis, which can sometimes be severe.

Discuss (6) | Improve

Next question

Aspergilloma

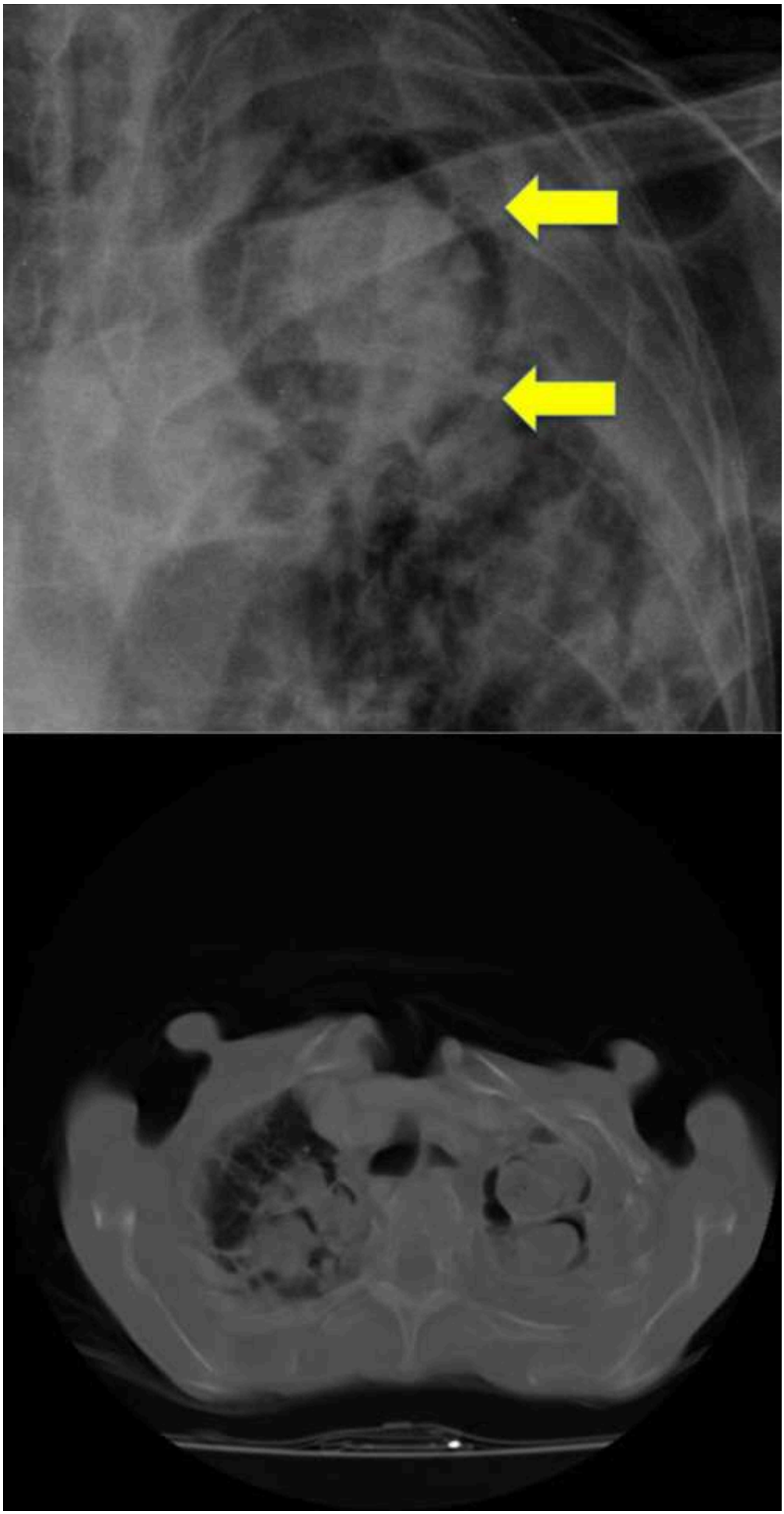
An aspergilloma is a mycetoma (mass-like fungus ball) which often colonises an existing lung cavity (e.g. secondary to tuberculosis, lung cancer or cystic fibrosis).

Usually asymptomatic but features may include

- cough
- haemoptysis (may be severe)

Investigations

- chest x-ray containing a rounded opacity. A crescent sign may be present
- high titres *Aspergillus* precipitins



© Image used on license from Radiopaedia

Aspergilloma in a patient with cavities secondary to previous tuberculosis infection. The close-up CXR and CT scan from the same patient demonstrate a rounded soft tissue attenuating masses located in a surrounding cavity.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **19%**

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Back to top

A 34 year-old man from South Korea presents with a two week history of abdominal pain in the right upper quadrant, fever and weight loss. The pain does not radiate anywhere and is 6/10 on the pain scale. Apart from the above he has no other symptoms. His only family history is his father who has hepatitis B and HIV. Examination reveals a 3cm tender palpable liver edge, pyrexia of 38.9ºC, pallor and right upper quadrant pain. There is no jaundice.

Blood tests reveal:

Bilirubin	29 µmol/l
ALP	200 u/l
ALT	90 u/l
γGT	100 u/l
Albumin	38 g/l

An ultrasound scan is requested and shows a 3x3cm abscess in the right lower lobe of the liver. The liver abscess is subsequently aspirated. Apart from aspiration of the large liver abscess, what is the most appropriate treatment?

- Blood cultures
- Karyotyping
- Metronidazole treatment
- Liver biopsy
- Mebendazole treatment

Submit answer

Reference ranges

Score: **16.7%**

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An ultrasound scan is requested and shows a 3x3cm abscess in the right lower lobe of the liver. The liver abscess is subsequently aspirated. Apart from aspiration of the large liver abscess, what is the most appropriate treatment?

Blood cultures	5%
Karyotyping	1%
Metronidazole treatment	71%
Liver biopsy	1%
Mebendazole treatment	22%

The most likely diagnosis in this case is an amoebic abscess, which are normally found in endemic tropical countries. Aspiration of large liver abscesses and a course of metronidazole is the preferred treatment for this condition.

Discuss (6) | Improve

Next question

Amoebiasis

Amoebiasis is caused by *Entamoeba histolytica* (an amoeboid protozoan) and spread by the faecal-oral route. It is estimated that 10% of the world's population is chronically infected. Infection can be asymptomatic, cause mild diarrhoea or severe amoebic dysentery. Amoebiasis also causes liver and colonic abscesses.

Amoebic dysentery

- profuse, bloody diarrhoea
- there may be a long incubation period
- stool microscopy may show trophozoites if examined within 15 minutes or kept warm (known as a 'hot stool')
- treatment
  - oral metronidazole
  - a 'luminal agent' (to eliminate intraluminal cysts) is recommended usually as well e.g. diloxanide furoate

Amoebic liver abscess

- usually a single mass in the right lobe (may be multiple). The contents are often described as 'anchovy sauce'
- features
  - fever
  - right upper quadrant pain
  - systemic symptoms e.g. malaise
  - hepatomegaly
- investigations
  - ultrasound
  - serology is positive in > 95%
- management
  - oral metronidazole
  - a 'luminal agent' (to eliminate intraluminal cysts) is recommended usually as well e.g. diloxanide furoate



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info

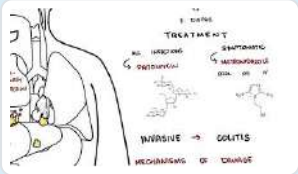
13 6

Amoebiasis review

Suggest link

Report broken link

Media



Amebiasis - entamoeba histolytica symptoms, pathophysiology, diagnosis, treatment

Armando Hasudungan - YouTube

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Report broken media

Score: **19%**

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Back to top

A 19 year-old University student presented to her General Practitioner with a three month history of intermittent diarrhoea and anorexia. She had lost approximately 10 kg of weight over a similar period. The patient had no previous abdominal symptoms and no significant past medical history. The symptoms had started several weeks after her return from a gap year expedition to Mali. On close questioning the patient did recall a short-lived intensely itchy erythematous rash on one of her feet while she was in Africa. This had spontaneously resolved and not subsequently recurred.

Initial investigations are listed below:

Hb	12.8 g/dl
Platelets	198 * 10^9/l
WBC	10.8 * 10^9/l
Neutrophils	5.2 * 10^9/l
Lymphocytes	2.1 * 10^9/l
Monocytes	0.8 * 10^9/l
Eosinophils	2.7 * 10^9/l
Basophils	0.0 * 10^9/l

Na^+	136 mmol/l
K^+	4.2 mmol/l
Urea	3.9 mmol/l
Creatinine	78 µmol/l

Stool microscopy: *Strongyloides stercoralis* larvae
Strongyloides stercoralis filariform larvae IgG: positive

Under supervision from Infectious Disease department the patient was treated with Ivermectin.

What is the best method to assess for *Strongyloides stercoralis* eradication six months after completion of treatment course?

- Repeat blood eosinophil count
- Assessment of patient symptoms
- Repeat *Strongyloides stercoralis* serology

- Repeat stool microscopy on two occasions one week apart
- Repeat stool microscopy with stool concentration technique

Submit answer

Reference ranges

Score: **16.7%**

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Strongyloides stercoralis filariform larvae IgG: positive

Under supervision from Infectious Disease department the patient was treated with Ivermectin.

What is the best method to assess for *Strongyloides stercoralis* eradication six months after completion of treatment course?

Repeat blood eosinophil count	7%
Assessment of patient symptoms	4%
Repeat *Strongyloides stercoralis* serology	23%

Repeat stool microscopy on two occasions one week apart	45%
Repeat stool microscopy with stool concentration technique	20%

Repeat serological testing is the best method of confirming *Strongyloides stercoralis* eradication following treatment. A post-treatment to pre-treatment titre ratio of < 0.6 is taken to represent a good indicator of treatment success.

Blood eosinophil count is also often found to decrease following treatment although this method clearly lacks specificity given the numerous other causes of eosinophilia.

Strongyloides stercoralis infection is often asymptomatic therefore resolution of symptoms does not provide good evidence of successful treatment. While stool microscopy is the gold standard diagnostic test for strongyloidiasis it has a sensitivity of only 50 % making it an unreliable tool to confirm eradication. Stool concentration techniques are used in specialist laboratories to improve sensitivity of stool microscopy for strongyloidiasis.

Greaves D, Coggle S, Pollard C, Aliyu S et al. Strongyloides stercoralis infection. BMJ 2013;347:4610.

Discuss (4) | Improve

Next question >

Strongyloides stercoralis

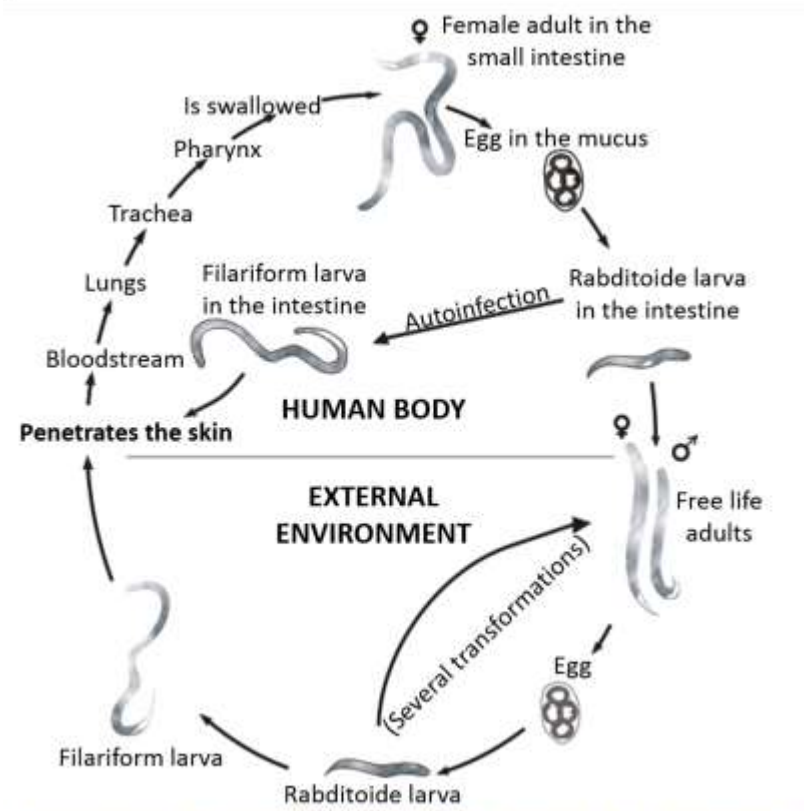
Strongyloides stercoralis is a human parasitic nematode worm. The larvae are present in soil and gain access to the body by penetrating the skin. Infection with *Strongyloides stercoralis* causes strongyloidiasis.

Features

- diarrhoea
- abdominal pain/bloating
- papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks
- larva currens: pruritic, linear, urticarial rash
- if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered

Treatment

- ivermectin and albendazole are used



Strongyloides stercoralis **Life Cycle**, Nematode (Hookworm)

Image sourced from Wikipedia

Diagram showing the lifecycle of *Strongyloides stercoralis*

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **19%**

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Back to top

A 22-year-old man presented to his General Practitioner with an itchy rash across his arms and legs. Symptoms had developed within the last few days and were causing the patient significant discomfort and preventing him from sleeping. The patient was a university student currently enjoying his summer vacation. He reported returning from a backpacking trip around Eastern Europe one week previously when he had stayed mostly in youth hostels. Since his return he had been staying in his parent's home. Interestingly, the patient had discovered on Facebook that a similar rash had afflicted a friend he had made at the end of his holiday.

He also worked part-time as a barman and denied coming into contact with any unusual chemicals at work. He had not recently introduced any new skin care products.

The patient had suffered from mild atopic eczema as an infant but had no recent skin complaints or other past medical history. He took no regular medications and had no known allergies. The patient smoked 10 cigarettes per day and drank roughly 40 units of alcohol each week.

On examination, the patient's arms and legs were covered with 2-5 mm maculopapular lesions with a central haemorrhagic punctum. In several places, several lesions were noted to be lying close together in a curve formation. There were minor marks of excoriation associated with the lesions. The patient's groin, abdomen, chest and back were free from rash. There was no axillary, cervical or inguinal lymphadenopathy.

What organism is the likely cause for the patient's rash?

- *Sarcoptes scabiei*
- *Leishmania donovani*
- *Ancylostoma braziliense*
- *Mycobacterium leprae*
- *Cimex hemipterus*

Submit answer

Reference ranges

Score: **16.7%**

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What organism is the likely cause for the patient's rash?

Sarcoptes scabiei	45%
Leishmania donovani	11%
Ancylostoma braziliense	16%
Mycobacterium leprae	3%
Cimex hemipterus	25%

The patient has symptoms and signs of exposure to bed bug infestation. In temperate regions this is caused by the insect *Cimex hemipteru* (or *Cimex lectularius* in tropical regions). A rash develops up to 11 days after exposure due to an allergic reaction. Bed bug infestation has increased in frequency over the past 10 years with the patient likely exposed during a recent stay at youth hostel.

The other organisms are all potential causes of skin disease in the returned traveller.

- *Sarcoptes scabiei* (scabies)
- *Leishmania donovani* (cutaneous leishmaniasis)
- *Ancylostoma braziliense* (cutaneous larva migrans)
- *Mycobacterium leprae*(leprosy)

Bernardeschi C, Le Cleach L, Delaunay P. Bed bug infestation. BMJ 2013;346:f138.

Discuss (10) Improve

Next question

Bed bugs

Bed bugs describes a variety of clinical problems including itchy skin rashes, bites and allergic symptoms secondary to infestation with *Cimex hemipteru*.

Bedbug infestation seems to be an increasingly common presentation within UK general practice. Bedbugs thrive in mattresses or fabrics, and can be extremely difficult to eradicate. Topical hydrocortisone is suitable to control itch, but definitive management is through a pest management company and fumigation of the house.

Bed bug numbers may be controlled by hot-washing bed linen and using mattress covers.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **19%**

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A 45-year-old female presents to your clinic with bilateral lower limb lesions. She said that it initially started as a small nodule on the right shin which then broke down into an exudative ulcer. Over the past year she has developed widespread 'cauliflower-like' lesions over her whole body. On examination you note a painless ulcer with scab over the anterior right shin, and generalised papillomas over the face, trunk, genitalia and buttocks.

Investigations demonstrate:

TPHA (Treponema pallidum Haem Agglutination test)	Positive

What is the most likely diagnosis?

- Pinta
- Syphilis
- Yaws
- Pyoderma gangrenosum
- Kaposi's sarcoma

Submit answer

Reference ranges

Score: **16.7%**

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Investigations demonstrate:

TPHA (Treponema pallidum Haem Agglutination test)	Positive

What is the most likely diagnosis?

Pinta	6%
Syphilis	42%
Yaws	43%
Pyoderma gangrenosum	4%
Kaposi's sarcoma	5%

Treponemal specific antibody tests (e.g. TPHA) are very specific to Treponema but not necessarily syphillis, as the species Treponema can cause a variety of diseases including syphilis, yaws and pinta

Important for me Less important

Care must be taken when interpreting syphilis investigations. Cardiolipin tests (e.g. VDRL and RPR) are non-specific and can be positive in many diseases such as TB, malaria and HIV. Treponemal specific antibody tests (e.g. TPHA) are very specific to Treponema, however the species Treponema can cause a variety of diseases including syphilis, yaws and pinta.

Yaws is a chronic infection that affects mainly the skin, bone and cartilage. The disease occurs mainly in low socio-economic communities in tropical areas of Africa, Asia and Latin America. The causative organism is a bacterium called Treponema pertenue, a subspecies of Treponema pallidum that causes venereal syphilis. However, yaws is a non-venereal infection.

In primary yaws, a single skin lesion develops at the point of entry of the bacterium after 2-4 weeks. This nodule can break down into an exudative ulcer. Without treatment, secondary yaws can occur, resulting in multiple lesions appear all over the body, more commonly over the face, trunk, genitalia and buttocks. Later on in the disease course, widespread bone, joint and soft tissue destruction can occur.

Discuss (9) | Improve

Next question >

Syphilis: investigation

Treponema pallidum is a very sensitive organism and cannot be grown on artificial media. The diagnosis is therefore usually based on clinical features, serology and microscopic examination of infected tissue.

Serological tests can be divided into:

- non-treponemal tests
  - not specific for syphilis, therefore may result in false positives (see below)
  - based upon the reactivity of serum from infected patients to a cardiolipin-cholesterol-lecithin antigen
  - assesses the quantity of antibodies being produced
  - becomes negative after treatment
  - examples include: rapid plasma reagin (RPR) and Venereal Disease Research Laboratory (VDRL)
- treponemal-specific tests
  - generally more complex and expensive but specific for syphilis
  - qualitative only and are reported as 'reactive' or 'non-reactive'
  - examples include: TP-EIA (*T. pallidum* enzyme immunoassay), TPHA (*T. pallidum* HaemAgglutination test)
  - the TP-EIA test has become increasingly popular in recent years

The testing algorithms for syphilis are complicated but typically involve a combination of a non-treponemal test with a treponemal-specific test.

Further points on non-treponemal tests

Causes of false positive non-treponemal (cardiolipin) tests:

- pregnancy
- SLE, anti-phospholipid syndrome
- tuberculosis
- leprosy
- malaria

- HIV

Example of test results

Positive non-treponemal test + positive treponemal test
- consistent with active syphilis infection

Positive non-treponemal test + negative treponemal test
- consistent with a false-positive syphilis result e.g. due to pregnancy or SLE (see list above)

Negative non-treponemal test + positive treponemal test :
- consistent with successfully treated syphilis



AI + Q 123

Next question >

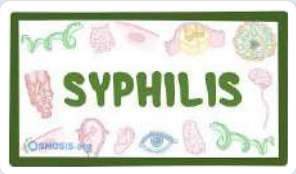
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Textbooks

High-yield textbook

Extended textbook

Media



Syphilis

Osmosis - YouTube 3 0

Report broken media

Score: **19%**

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A 25-year-old HIV-positive male goes to his GP with complaints of headache and left-sided weakness of recent onset. His temperature is 38ºC, blood pressure is 115/70 mmHg, respirations are 14/min and pulse is 73/min.

Neurological examination reveals decreased power, hyperreflexia in the left upper and lower limb with associated upgoing plantars. CT head shows multiple ring-enhancing lesions.

What is the most appropriate next step in management?

- ○ Trimethoprim-sulfamethoxazole ×
- ○ Sulfadiazine and pyrimethamine ×
- ○ Brain irradiation ×
- ○ Brain biopsy ×
- ○ Start albendazole ×

Submit answer

Reference ranges

Score: **16.7%**

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Back to top

A 25-year-old HIV-positive male goes to his GP with complaints of headache and left-sided weakness of recent onset. His temperature is 38ºC, blood pressure is 115/70 mmHg, respirations are 14/min and pulse is 73/min.

Neurological examination reveals decreased power, hyperreflexia in the left upper and lower limb with associated upgoing plantars. CT head shows multiple ring-enhancing lesions.

What is the most appropriate next step in management?

Trimethoprim-sulfamethoxazole	7%
Sulfadiazine and pyrimethamine	91%
Brain irradiation	1%
Brain biopsy	1%
Start albendazole	1%

HIV, neuro symptoms, multiple brain lesions with ring enhancement - toxoplasmosis

Important for me Less important

The most common cause of central nervous system mass lesion in AIDS patients is toxoplasmosis. This patient's findings are consistent with cerebral toxoplasmosis. Trimethoprim-sulfamethoxazole is used for prophylaxis of toxoplasmosis, while sulfadiazine and pyrimethamine are used for treatment purposes.

Choice 3: Brain irradiation is used in the management of primary CNS lymphoma. Patients with primary CNS lymphoma are usually afebrile; lesions are weakly enhancing and are single (can be multiple). This is suspected if the patient does not respond to antibiotic therapy.

Choice 4: Brain biopsy is reserved for patient's whose lesions do not respond to treatment with sulfadiazine and pyrimethamine.

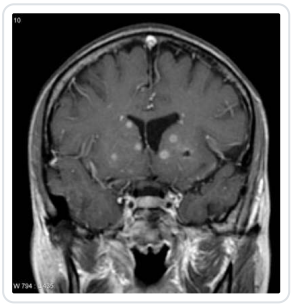
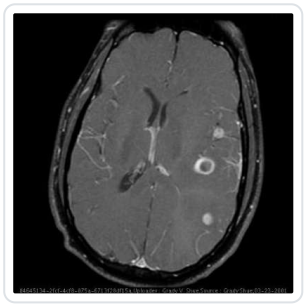
Choice 5: Albendazole is used for neurocysticercosis. This condition is not usually seen in AIDS patients.

HIV: neurocomplications ★

Focal neurological lesions

Toxoplasmosis

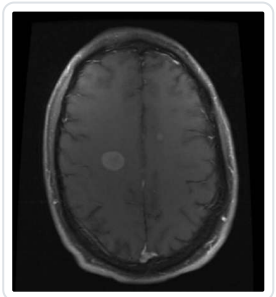
- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	Lymphoma
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination
- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link

Report broken link

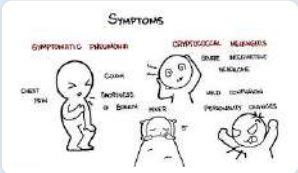
Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

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Report broken media

Score: **19%**

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Back to top

A 44-year-old man is reviewed in the renal clinic. He has a diagnosis of end-stage renal failure due to IgA nephropathy. The team is working him up for a renal transplant.

As part of his transplant workup, he has an interferon-gamma release assay performed:

Interferon-gamma release assay	Positive

He subsequently has further testing:

Nucleic acid amplification tests	Negative for *Mycobacterium tuberculosis*.

Sputum acid-fast bacilli smear	Negative for acid-fast bacilli

Chest X-ray	Lung fields appear clear. Normal cardiomediastinal contour.

What is the most appropriate management?

- No treatment required
- Pyridoxine for 6 months
- Rifampicin and isoniazid (with pyridoxine) for 3 months
- Rifampicin, isoniazid (with pyridoxine), pyrazinamide, and ethambutol for 2 months, then rifampicin and isoniazid for 10 months
- Rifampicin, isoniazid (with pyridoxine), pyrazinamide, and ethambutol for 2 months, then rifampicin and isoniazid for 2 months

Submit answer

Reference ranges

Score: **16.7%**

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As part of his transplant workup, he has an interferon-gamma release assay performed:

Interferon-gamma release assay	Positive

He subsequently has further testing:

Nucleic acid amplification tests	Negative for *Mycobacterium tuberculosis*.

Sputum acid-fast bacilli smear	Negative for acid-fast bacilli

Chest X-ray	Lung fields appear clear. Normal cardiomediastinal contour.

What is the most appropriate management?

No treatment required	21%
Pyridoxine for 6 months	2%
Rifampicin and isoniazid (with pyridoxine) for 3 months	72%
Rifampicin, isoniazid (with pyridoxine), pyrazinamide, and ethambutol for 2 months, then rifampicin and isoniazid for 10 months	2%
Rifampicin, isoniazid (with pyridoxine), pyrazinamide, and ethambutol for 2 months, then rifampicin and isoniazid for 2 months	3%

Positive IGRA, but asymptomatic and normal CXR → treat as for latent tuberculosis

Important for me Less important

Rifampicin and isoniazid (with pyridoxine) for 3 months is correct. This patient has latent *Mycobacterium tuberculosis* infection: he has a positive interferon-gamma release assay but negative sputum for acid-fast bacilli and nucleic acid amplification tests as well as a normal chest x-ray. Latent *Mycobacterium tuberculosis* infection can either be treated with 3 months of rifampicin and isoniazid or with 6 months of isoniazid.

No treatment required is incorrect. This patient has a positive interferon-gamma release assay. This suggests either active or latent *Mycobacterium tuberculosis* infection. Given the negative nucleic acid amplification test, sputum smear for acid-fast bacilli, and normal chest x-ray, it is likely this patient has latent *Mycobacterium tuberculosis* infection. This requires treatment with either 3 months of rifampicin and isoniazid or 6 months of isoniazid alone.

Pyridoxine for 6 months is incorrect. Pyridoxine is a form of vitamin B6 and is used for the prevention of isoniazid-induced neuropathy. It is not in itself used as part of the treatment of *Mycobacterium tuberculosis* infection.

Rifampicin, isoniazid (with pyridoxine), pyrazinamide, and ethambutol for 2 months, then rifampicin and isoniazid for 10 months is incorrect. This is the treatment regimen for active *Mycobacterium tuberculosis* infection with CNS involvement. It is also usually accompanied by a course of steroids. The patient in this case, however, has latent *Mycobacterium tuberculosis* infection, evidenced by the positive interferon-gamma release assay but negative sputum for acid-fast bacilli and nucleic acid amplification tests as well as a normal chest x-ray.

Rifampicin, isoniazid (with pyridoxine), pyrazinamide, and ethambutol for 2 months, then rifampicin and isoniazid for 4 months is incorrect. This is the treatment regimen for active *Mycobacterium tuberculosis* infection without CNS involvement. The patient in this question has latent *Mycobacterium tuberculosis* infection, so this answer is incorrect.

Discuss (1) | Improve

Next question ›

Tuberculosis: latent disease ★

Patients with latent tuberculosis are asymptomatic and non-infectious. They are typically diagnosed by having a positive tuberculin skin test or Interferon-Gamma Release Assay (IGRA) combined with a normal chest x-ray, helping to exclude active tuberculosis.

NICE now give two choices for treating **latent tuberculosis**:

- 3 months of isoniazid (with pyridoxine) and rifampicin, or
- 6 months of isoniazid (with pyridoxine)

Why two choices? NICE (2016) state the following:

> *Base the choice of regimen on the person's clinical circumstances. Offer:*

- *3 months of isoniazid (with pyridoxine) and rifampicin to people younger than 35 years if hepatotoxicity is a concern after an assessment of both liver function (including transaminase levels) and risk factors*
- *6 months of isoniazid (with pyridoxine) if interactions with rifamycins are a concern, for example, in people with HIV or who have had a transplant.*

People with latent tuberculosis cannot pass the disease on to others, so there is no restriction in terms of employment etc. This advice would obviously change if the patient developed active tuberculosis at any point.

Risk factors for developing active tuberculosis include:

- silicosis
- chronic renal failure
- HIV positive
- solid organ transplantation with immunosuppression
- intravenous drug use
- haematological malignancy
- anti-TNF treatment
- previous gastrectomy



Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2016 Tuberculosis guidelines

1 0

Greenbook 0 0

Tuberculosis

Suggest link Report broken link

Media



Tuberculosis

Osmosis - YouTube 2 0

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Score: **19%**

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A 48 year-old civil servant was referred to the Acute Medical Unit by his general practitioner with progressive cognitive impairment and severe lethargy over the last 3 weeks. He had also become intermittently agitated and his sleep was disturbed during the night. He had returned from visiting his partners relatives in Zimbabwe 1 month ago. He suffered from type II diabetes which was well controlled through dietary modification. He smoked 10 cigarettes a day and drank 14 units of alcohol per week.

On examination, his temperature was 37.2ºC, pulse was 60 beats per minute and blood pressure was 134/88. His Glasgow Coma Score was 14/15 due to confused speech. His trachea was central and right-sided posterior cervical lymphadenopathy was present. His jugular venous pressure was not elevated and chest was clear on auscultation. Heart sounds were normal.

Investigations:

Haemoglobin	143 g/L (130-180)
White cell count	7.7 x 109/L (4.0-11.0)
Neutrophil count	3.0 x 109/L (2.0-7.5)
Lymphocyte count	1.8 x 109/L (1.3-3.5)
Eosinophil count	0.4 X 109/L (0.1-0.4)
Platelets	310 x 109/L (150-400)

Sodium	134 mmol/L (135-145)
Potassium	4.8 mmol/L (3.5-5.0)
Urea	9.0 mmol/L (2.5-7.5)
Creatinine	100 mol/L (25-95)
Fasting plasma glucose	4.4 mmol/L (3.0-6.0)

CT head	No abnormality detected

What is the most appropriate next investigation?

- MRI brain
- Lymph node biopsy
- Lumbar puncture

- [ ] Stool microscopy
- [ ] Blood film

Submit answer

Reference ranges

Score: **16.7%**

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Creatinine	100 mol/L (25-95)
Fasting plasma glucose	4.4 mmol/L (3.0-6.0)

CT head	No abnormality detected

What is the most appropriate next investigation?

MRI brain	10%
Lymph node biopsy	24%
Lumbar puncture	31%

Stool microscopy	7%
Blood film	27%

This patient has African trypanosomiasis, sometimes known as sleeping sickness. The acute presentation and country of origin suggest the East African variant, caused by *Trypanosoma bruicei rhodesiense*. Although the parasites may be detected in a peripheral blood film or lymph node biopsy, determining whether they have penetrated the central nervous system (CNS) will most influence choice of drug therapy. Pentamidine is the treatment of choice in early infection, while choices for CNS infection include suramin, melarsoprol, eflornithine and nifurtimox. These medications are not widely available and can result in extremely severe side effects, so are only prescribed by tropical medicine specialists.

Discuss (12) Improve

Next question

Trypanosomiasis

Two main form of this protozoal disease are recognised - African trypanosomiasis (sleeping sickness) and American trypanosomiasis (Chagas' disease).

Two forms of **African trypanosomiasis**, or **sleeping sickness**, are seen - *Trypanosoma gambiense* in West Africa and *Trypanosoma rhodesiense* in East Africa. Both types are spread by the tsetse fly. *Trypanosoma rhodesiense* tends to follow a more acute course. Clinical features include:

- Trypanosoma chancre - painless subcutaneous nodule at site of infection
- intermittent fever
- enlargement of posterior cervical lymph nodes
- later: central nervous system involvement e.g. somnolence, headaches, mood changes, meningoencephalitis

Management

- early disease: IV pentamidine or suramin
- later disease or central nervous system involvement: IV melarsoprol

American trypanosomiasis, or **Chagas' disease**, is caused by the protozoan *Trypanosoma cruzi*. The vast majority of patients (95%) are asymptomatic in the acute phase although a chagoma (an erythematous nodule at site of infection) and periorbital oedema are sometimes seen. Chronic Chagas' disease mainly affects the heart and gastrointestinal tract

- myocarditis may lead to dilated cardiomyopathy (with apical atophy) and arrhythmias

- gastrointestinal features includes megaoesophagus and megacolon causing dysphagia and constipation

Management

- treatment is most effective in the acute phase using azole or nitroderivatives such as benznidazole or nifurtimox
- chronic disease management involves treating the complications e.g., heart failure



Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

8 3

Chaga's disease

Suggest link Report broken link

Media



Sleeping Sickness - an introduction to African Trypanosomiasis

Let's Learn About Bugs - YouTube 7 0



Chagas Disease - An overview

Let's Learn About Bugs - YouTube 3 1



Chagas disease

Osmosis - YouTube 2 1

Report broken media

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Back to top

A 33-year-old man who has recently emigrated from Zimbabwe is admitted after being found confused at home. His partner reports that he has been acting strangely for a number of weeks and complaining of altered taste and dizziness. She reports that she is HIV positive but her partner refuses to be tested as he does not believe in 'medical lies'.

On admission he is apyrexial with a blood pressure of 114/82mmHg and pulse 78/min.

Rapid HIV testing confirms that he has the infection. His CD4 count is 11 cells/µl.

An MRI (T1 C+) shows the following:



© Image used on license from Radiopaedia

Other than starting highly active antiretroviral treatment, what is the most appropriate treatment?

- Amphotericin B
- Surgical resection
- Steroids + methotrexate + whole brain irradiation
- Rifampicin + isoniazid + pyrazinamide + ethambutol + steroids

Sulfadiazine + pyrimethamine

Submit answer

Reference ranges

Score: **16.7%**

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On admission he is apyrexial with a blood pressure of 114/82mmHg and pulse 78/min.

Rapid HIV testing confirms that he has the infection. His CD4 count is 11 cells/µl.

An MRI (T1 C+) shows the following:



© Image used on license from Radiopaedia

Other than starting highly active antiretroviral treatment, what is the most appropriate treatment?

Amphotericin B	6%
Surgical resection	0%
Steroids + methotrexate + whole brain irradiation	8%
Rifampicin + isoniazid + pyrazinamide + ethambutol + steroids	6%

Sulfadiazine + pyrimethamine 81%

HIV, neuro symptoms, multiple brain lesions with ring enhancement - toxoplasmosis

Important for me Less important

The correct answer in this case is **sulfadiazine + pyrimethamine**. The patient's presentation with confusion, altered taste, dizziness, and a low CD4 count of 11 cells/µl suggests that he has an opportunistic infection due to his HIV infection. The MRI image provided shows multiple ring-enhancing lesions, which are consistent with cerebral toxoplasmosis. Cerebral toxoplasmosis is caused by the protozoan parasite *Toxoplasma gondii* and is a common opportunistic infection in patients with advanced HIV disease. The recommended treatment for cerebral toxoplasmosis is a combination of sulfadiazine and pyrimethamine.

Amphotericin B is an antifungal medication used primarily to treat invasive fungal infections such as cryptococcal meningitis or disseminated histoplasmosis. Although these infections can occur in patients with HIV, the MRI findings and clinical presentation are more suggestive of cerebral toxoplasmosis.

Surgical resection might be considered for space-occupying lesions such as brain abscesses or tumours. However, given the patient's multiple ring-enhancing lesions on MRI and low CD4 count, cerebral toxoplasmosis is much more likely than a primary brain tumour or abscesses.

Steroids + methotrexate + whole brain irradiation would be a treatment approach for primary central nervous system (CNS) lymphoma, which can also present with neurological symptoms in patients with HIV. However, CNS lymphoma typically presents as a solitary lesion rather than multiple ring-enhancing lesions seen in this case.

Rifampicin + isoniazid + pyrazinamide + ethambutol + steroids represent the standard regimen for treating tuberculosis (TB). While TB can involve the CNS in patients with HIV, the MRI findings are not typical for CNS TB. Moreover, the patient's symptoms of altered taste and dizziness do not fit well with a diagnosis of CNS TB.

Discuss (2) Improve

Next question ›

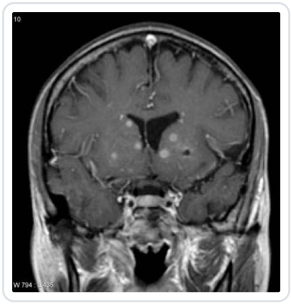
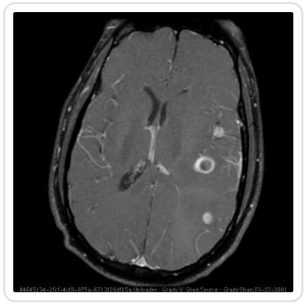
HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

- accounts for around 50% of cerebral lesions in patients with HIV

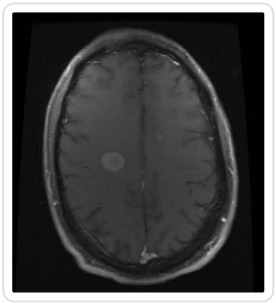
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination
- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link

Report broken link

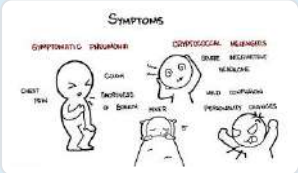
Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

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Report broken media

Score: **19%**

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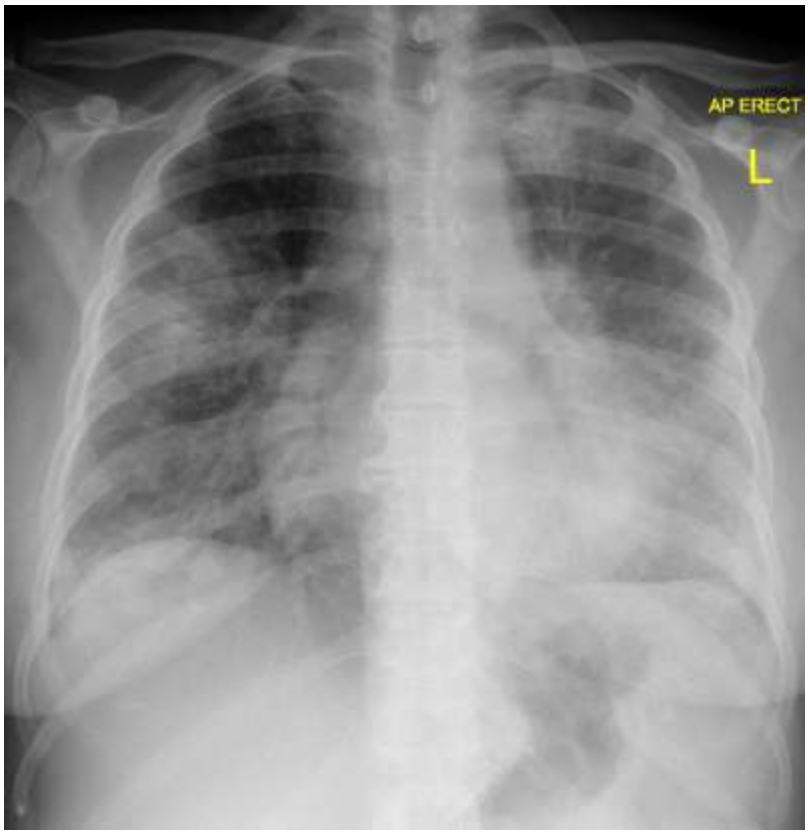
A 54-year-old woman presents to the Acute Medical Unit with a three day history of fever, myalgia and a non-productive cough. She initially thought her symptoms were due to the 'flu but over the past 24 hours has been feeling progressively more poorly. She now also has nausea, abdominal pain, headaches and diarrhoea.

On examination her pulse is 84/min, temperature 39.4ºC and blood pressure 106/76 mmHg. Bilateral scattered crackles are noted on auscultation.

Bloods show the following:

Hb	12.4 g/l	Na^+	133 mmol/l
Platelets	363 * 10^9/l	K^+	5.0 mmol/l
WBC	12.1 * 10^9/l	Urea	7.8 mmol/l
Neuts	10.8 * 10^9/l	Creatinine	88 µmol/l
Lymphs	1.1 * 10^9/l	CRP	145 mg/l
Eosin	0.2 * 10^9/l		

The chest x-ray is shown below:



© Image used on license from Radiopaedia

Which investigation is most likely to be diagnostic?

- Sputum culture
- Cold agglutinins
- Bronchoalveolar lavage
- Blood serology
- Urinary antigen

Submit answer

Reference ranges

Score: **0%**

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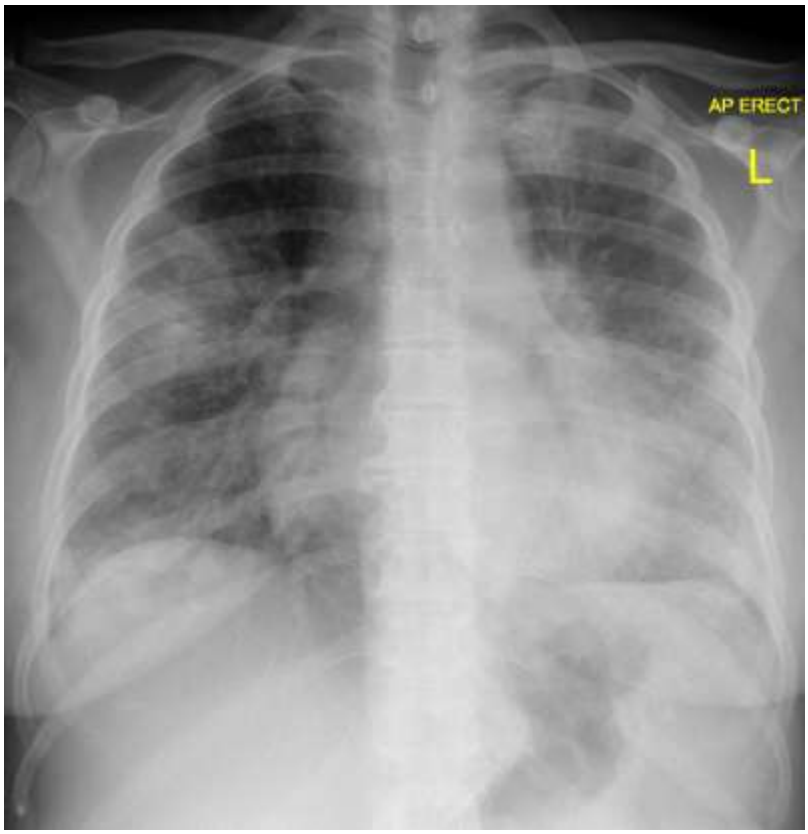
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Neuts	10.8 * 10^9/l	Creatinine	88 µmol/l
Lymphs	1.1 * 10^9/l	CRP	145 mg/l
Eosin	0.2 * 10^9/l		

The chest x-ray is shown below:



© Image used on license from Radiopaedia

Which investigation is most likely to be diagnostic?

Sputum culture	5%
Cold agglutinins	10%
Bronchoalveolar lavage	10%
Blood serology	13%
Urinary antigen	**62%**

Legionella pneumophilia is best diagnosed by the **urinary antigen** test

Important for me Less important

The chest x-ray shows the asymmetrical, patchy bilateral consolidation typical of *Legionella*. This combined with high fever, hyponatraemia, abdominal pain and lymphopaenia make the diagnosis the most likely.

Discuss (10) Improve

Next question >

Legionella

Legionnaire's disease is caused by the intracellular bacterium *Legionella pneumophilia*. It typically colonizes water tanks and hence questions may hint at air-conditioning systems or foreign holidays. Person-to-person transmission is not seen

Features

- flu-like symptoms including fever (present in > 95% of patients)
- dry cough
- relative bradycardia
- confusion
- lymphopaenia
- hyponatraemia
- deranged liver function tests
- pleural effusion: seen in around 30% of patients

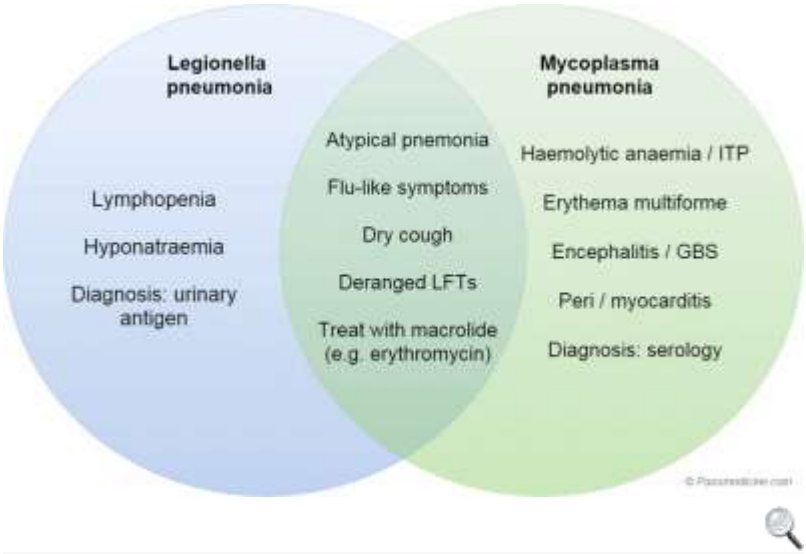
Investigations

- diagnositic test of choice:urinary antigen

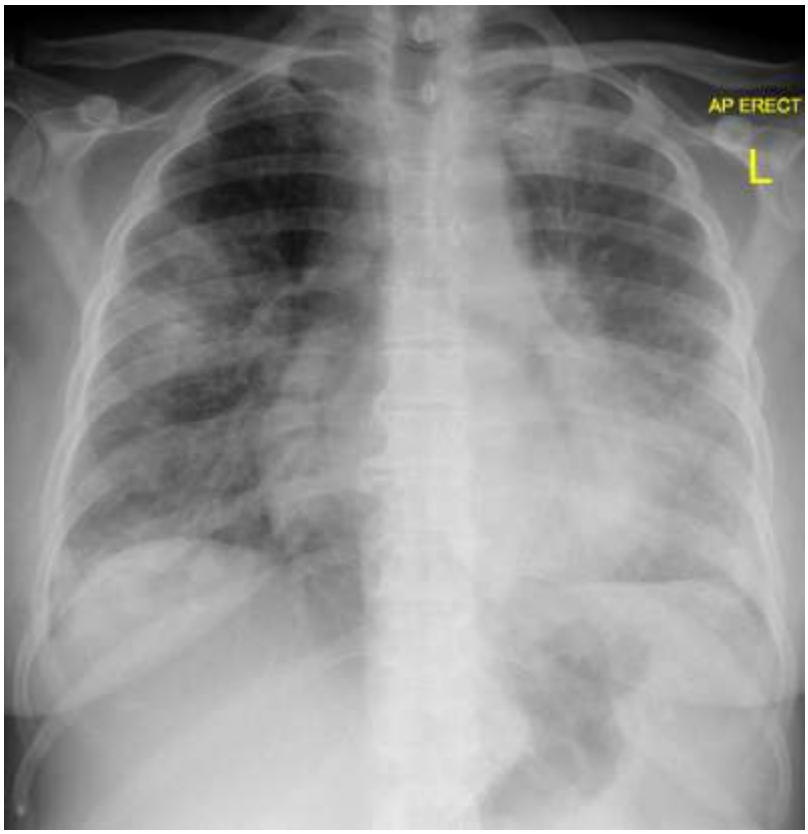
- chest x-ray findings are non-specific but may include:
  - a mid-to-lower zone predominance of patchy consolidation
  - pleural effusions in around 30%

Management

- treat with erythromycin/clarithromycin



Comparison of Legionella and Mycoplasma pneumonia



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Chest x-ray features of legionella pnuemonia are non-specific but includes a mid-to-lower zone predominance of patchy consolidation. Pleural effusions are seen in around 30%.

Next question

Textbooks

High-yield textbook

Extended textbook

Media



Legionella pneumophila

Osmosis - YouTube 4 0

Report broken media

Score: **32%**

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A 17-year-old man who has recently come to the UK from Rwanda is admitted to hospital. His friend describes him complaining of a headache and fever for the past four days. Further history reveals that he is very lethargic with a dry cough and generalised myalgia. The patient also describes passing some dark urine this morning. He has no past medical history of note. On examination his pulse is 110/min, temperature 38.1°C, oxygen saturations 98% on room air and blood pressure 110/68 mmHg, His sclera are jaundiced and there is enlargement of the liver and spleen. Bloods show the following:

Na^+	142 mmol/l
K^+	4.8 mmol/l
Urea	12.3 mmol/l
Creatinine	144 µmol/l

What is the most likely diagnosis?

- Typhoid
- Dengue fever
- African trypanosomiasis
- Malaria
- Leishmaniasis

Submit answer

Reference ranges

Score: **0%**

1 -
2 -

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Back to top

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Na^+	142 mmol/l
K^+	4.8 mmol/l
Urea	12.3 mmol/l
Creatinine	144 µmol/l

What is the most likely diagnosis?

Typhoid	5%
Dengue fever	17%
African trypanosomiasis	15%
Malaria	48%
Leishmaniasis	15%

This patient clearly has an infective process so much of the data (headache, fever, myalgia, vital signs) can be discarded as being common to all the answers.

We're now left with more concrete findings: jaundice, hepatosplenomegaly and a raised creatinine.

Now we take one of these findings, hepatosplenomegaly and consider the differential diagnosis:

Causes of hepatosplenomegaly

- chronic liver disease with portal hypertension
- infections: glandular fever, malaria, hepatitis
- lymphoproliferative disorders
- myeloproliferative disorders e.g. chronic myeloid leukaemia
- amyloidosis

This suggests that malaria is the most likely diagnosis. Jaundice and a raised creatinine are consistent with this.

Discuss (8) | Improve

Next question >

Malaria: Falciparum

Falciparum malaria is the commonest, and most severe, type of malaria.

Clinical features

Falciparum malaria typically presents with symptoms suggestive of a flu-like illness. The classical triad of symptoms includes paroxysms of fever, chills, and sweating. These symptoms may occur every 48 hours corresponding to the erythrocytic cycle of the *Plasmodium falciparum* parasite. Fever is often high, intermittent, and may be accompanied by rigors. Initial manifestations can be non-specific and include malaise, headache, and myalgia, which might be mistaken for a viral syndrome.

General features of falciparum malaria

- fever: The hallmark of malaria, typically cyclical, often accompanied by sweating and sometimes rigors.
- gastrointestinal:
  - anorexia, nausea, vomiting, and abdominal pain are frequent
  - diarrhoea can occasionally be a feature, more commonly in children
  - patients may exhibit mild jaundice and occasional pruritus
- respiratory: Cough and, in some cases, mild tachypnoea may occur, though primary respiratory complications are rare in non-severe cases.
- musculoskeletal: Generalised body aches and joint pains are common.
- neurological:
  - headache is prominent and often severe.
  - dizziness and sleep disturbances may also be observed.
- cardiovascular: Tachycardia may be evident, and although hypotension is more typical of severe malaria, it can occasionally be seen in non-severe cases as well.
- haematological:
  - thrombocytopaenia is the most significant haematological finding and can occur in the absence of severe disease.
  - mild anaemia may also be present.
- renal: While acute kidney injury is associated with severe malaria, non-severe cases might show mild to moderate increases in creatinine or blood urea nitrogen levels.

Feature of severe falciparum malaria

- schizonts on a blood film
- parasitaemia > 2%
- hypoglycaemia
- acidosis
- temperature > 39 °C
- severe anaemia
- complications as below

Complications

- cerebral malaria: seizures, coma
- acute renal failure: blackwater fever, secondary to intravascular haemolysis, mechanism unknown
- acute respiratory distress syndrome (ARDS)
- hypoglycaemia
- disseminated intravascular coagulation (DIC)

Management

Uncomplicated falciparum malaria

- strains resistant to chloroquine are prevalent in certain areas of Asia and Africa
- the 2010 WHO guidelines recommend artemisinin-based combination therapies (ACTs) as first-line therapy
- examples include artemether plus lumefantrine, artesunate plus amodiaquine, artesunate plus mefloquine, artesunate plus sulfadoxine-pyrimethamine, dihydroartemisinin plus piperaquine

Severe falciparum malaria

- a parasite counts of more than 2% will usually need parenteral treatment irrespective of clinical state
- intravenous artesunate is now recommended by WHO in preference to intravenous quinine
- if parasite count > 10% then exchange transfusion should be considered
- shock may indicate coexistent bacterial septicaemia - malaria rarely causes haemodynamic collapse

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

WHO

9 7

Malaria treatment guidelines

Suggest link

Report broken link

Media



Malaria

Osmosis - YouTube

5 1

Report broken media

Score: **32%**

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Back to top

A 23-year-old woman presents to the emergency department. She was brought in by ambulance following left-sided weakness and difficulty in finding words. She also complains of a headache and nausea for the last three days. She has no past medical history and does not smoke nor does she drink alcohol. She recently emigrated from Tanzania to the UK.

On examination, she has marked weakness in upper and lower left limbs and is unable to walk without assistance. Her responses to questions are slow and limited.

Blood tests:

Hb	129 g/l
Platelets	316 * 10^9/l
WBC	8.9 * 10^9/l
Na^+	141 mmol/l
K^+	4.7 mmol/l
Urea	5.1 mmol/l
Creatinine	71 µmol/l
HIV-1 serology	positive
HIV-2 serology	negative
HIV viral load	pending
CD4 count	pending
Toxoplasmosis serology	pending
Cryptococcal antigen	pending

A CT scan of her head demonstrates multiple ring-enhancing lesions and mass effect. Dexamethasone is started immediately. What is the most appropriate next step?

- Antiretroviral treatment
- Amphotericin
- Methotrexate
- MRI brain and whole spine
- Pyrimethamine and sulfadiazine

Submit answer

Reference ranges

Score: **0%**

1 -
2 -
3 -

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HIV viral load	pending
CD4 count	pending
Toxoplasmosis serology	pending
Cryptococcal antigen	pending

A CT scan of her head demonstrates multiple ring-enhancing lesions and mass effect. Dexamethasone is started immediately. What is the most appropriate next step?

Antiretroviral treatment	6%
Amphotericin	2%
Methotrexate	1%
MRI brain and whole spine	1%
Pyrimethamine and sulfadiazine	**90%**

HIV, neuro symptoms, multiple brain lesions with ring enhancement - toxoplasmosis

Important for me Less important

The correct answer is pyrimethamine and sulfadiazine as the patient likely has cerebral toxoplasmosis as an AIDS-defining illness. Methotrexate is the treatment for a CNS lymphoma, alongside whole brain radiotherapy, but CNS lymphoma is unlikely since there are multiple lesions. Antiretroviral treatment will need to be started but should be done in specialist settings and not in the acute one. Further imaging would not be needed as there are sufficient evidence to start treatment at this point and would cause an unnecessary delay.

Discuss (3) Improve

Next question >

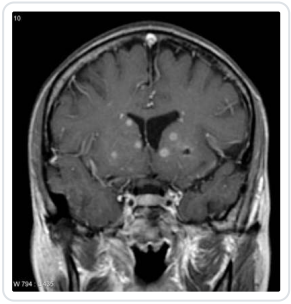
HIV: neurocomplications

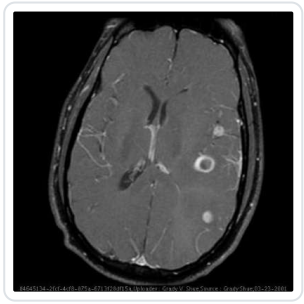
Focal neurological lesions

Toxoplasmosis

- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine



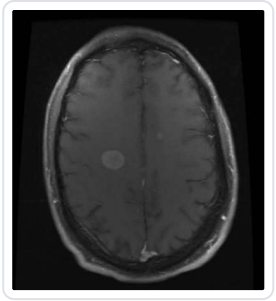




Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination

- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy



Next question

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Links

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Toxoplasmosis vs lymphoma

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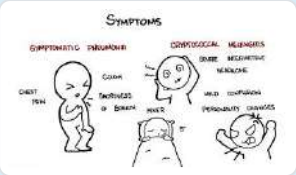
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Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

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Cryptococcus neoformans infection

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A 36 year old male Intravenous drug user presents to genito-urinary clinic. He was diagnosed with HIV 3 years ago after presenting with tuberculosis (TB). He was treated for TB for 6 months. He is now on third line anti-retroviral therapy for previous virological failure and co-trimoxazole. His most recent CD4 count was 104, and his viral load was 3,000 copies/ml.

He complains of weakness on his left side, and deterioration in vision, getting worse for four weeks. He has a moderately severe headache. On examination his visual acuity is 3/60 in his left eye and 6/6 in his right eye. He has weakness, in his left arm and leg, brisk reflexes and mildly increased tone.

He is immediately admitted.

A CT scan shows several ring enhancing lesions in his right cerebral hemispheres and one on the left cerebral hemisphere.

On ophthalmological review he has a large area of retinal necrosis in his left eye.

What is the diagnosis?

- CNS lymphoma
- Cerebral toxoplasmosis
- Cerebral TB
- Cerebral abscesses
- CNS Kaposi sarcoma

Submit answer

Reference ranges

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He is immediately admitted.

A CT scan shows several ring enhancing lesions in his right cerebral hemispheres and one on the left cerebral hemisphere.

On ophthalmological review he has a large area of retinal necrosis in his left eye.

What is the diagnosis?

CNS lymphoma	2%
Cerebral toxoplasmosis	93%
Cerebral TB	2%
Cerebral abscesses	1%
CNS Kaposi sarcoma	1%

HIV, neuro symptoms, multiple brain lesions with ring enhancement - toxoplasmosis

Important for me Less important

The most important three differential diagnosis for a ring-enhancing lesion in an HIV patient is CNS lymphoma, TB and Toxoplasmosis.

In this case the retinal necrosis in characteristic of ophthalmic toxomplasmosis. Multiple lesion also favour toxoplasmosis. His poor compliance with HIV treatment (as indicated by the low CD4 count

and the high viral load) suggest his compliance with co-trimoxazole is also poor.

Discuss (5) | Improve

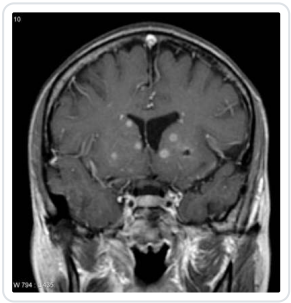
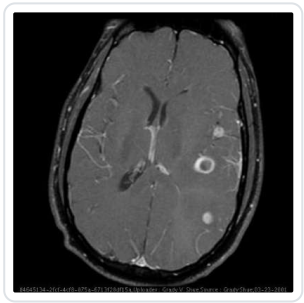
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HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

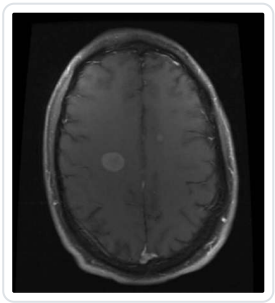
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Toxoplasmosis vs lymphoma

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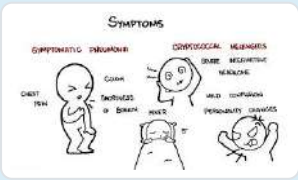
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Cryptococcus neoformans infection

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A 68-year-old man presents to hospital with progressive shortness of breath over the last four days and a low-grade fever. He has a past medical history of mild asthma for which he occasionally needs to use his salbutamol inhaler and has previously had bilateral knee replacements for osteoarthritis. He has smoked on average 5 cigarettes per day for the past 40 years and drinks a couple of glasses of wine per week. His travel history includes a holiday to Cyprus, from which he arrived back in the UK 5 days ago.

Examination revealed some right mid zone crackles and reduced breath sounds over this area. Observations revealed a temperature of 38.8ºC, heart rate of 110 bpm, blood pressure of 105/66 mmHg, respiratory rate of 22 breaths per minute and oxygen saturations of 91% on room air.

Blood tests revealed:

Hb	145 g/l
Platelets	290 * 10^9/l
WBC	9.4 * 10^9/l
Na^+	132 mmol/l
K^+	3.7 mmol/l
Urea	4.1 mmol/l
Creatinine	67 µmol/l

Urinary sodium concentration was measured and found to be 36mmol/L (normal range 40-220 mmol/d). Which of the following investigations is most useful in the diagnosis of this condition?

- Urine antigen test
- Serum antibody test
- Sputum PCR
- CT pulmonary angiography
- D-dimer

Submit answer

Reference ranges

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Creatinine	67 µmol/l

Urinary sodium concentration was measured and found to be 36mmol/L (normal range 40-220 mmol/d). Which of the following investigations is most useful in the diagnosis of this condition?

Urine antigen test	91%
Serum antibody test	3%
Sputum PCR	4%
CT pulmonary angiography	2%
D-dimer	0%

Legionella pneumophilia is best diagnosed by the **urinary antigen** test

Important for me Less important

This scenario is most likely due to the organism *Legionella pneumophila* (causing Legionnaire's disease) - an infection which is often due to air conditioning units and heating units. This patient may have got the infection when on holiday and his sodium is low, often a result of syndrome of inappropriate ADH secretion. The most accurate investigation for Legionella infection is via the urine antigen test, which is quick and specific.

Discuss (4) Improve

Next question

Legionella

Legionnaire's disease is caused by the intracellular bacterium *Legionella pneumophilia*. It typically colonizes water tanks and hence questions may hint at air-conditioning systems or foreign holidays. Person-to-person transmission is not seen

Features

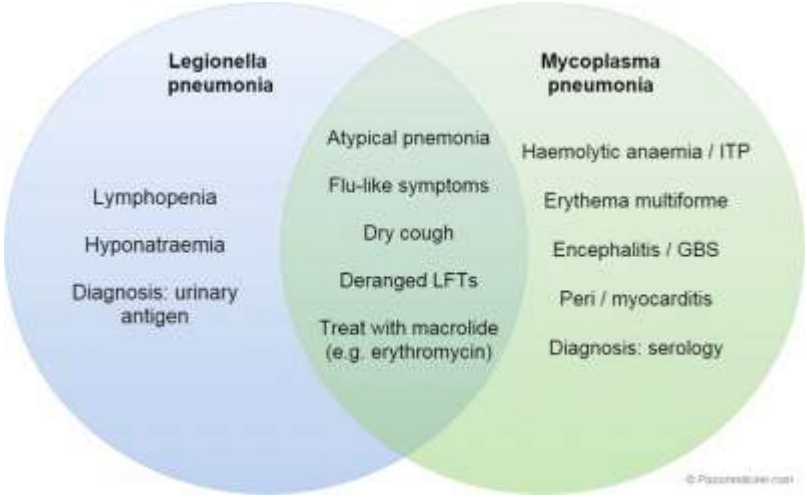
- flu-like symptoms including fever (present in > 95% of patients)
- dry cough
- relative bradycardia
- confusion
- lymphopaenia
- hyponatraemia
- deranged liver function tests
- pleural effusion: seen in around 30% of patients

Investigations

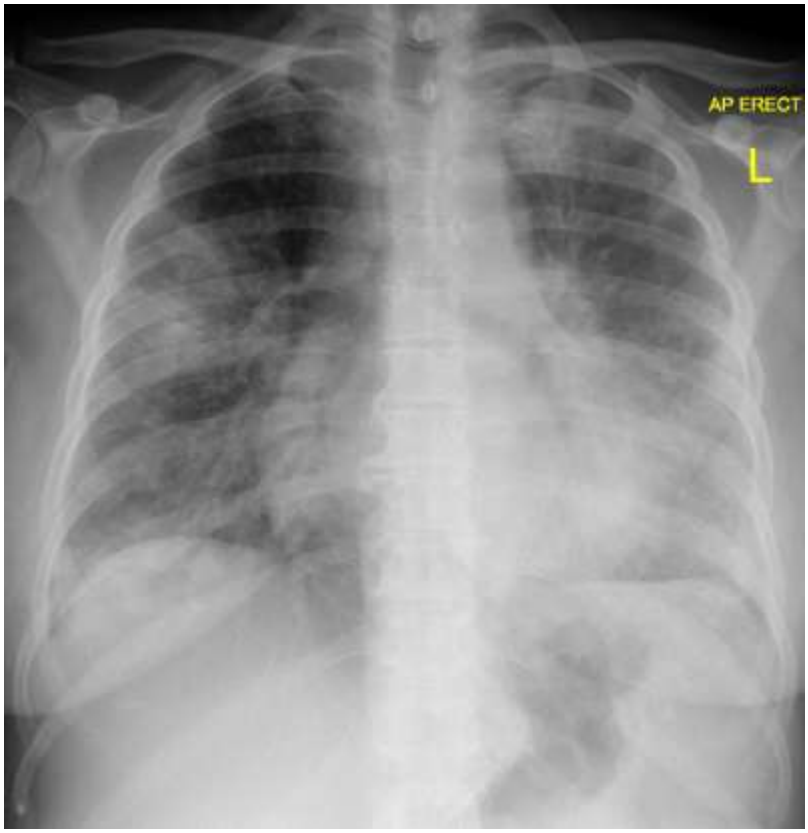
- diagnositic test of choice:urinary antigen
- chest x-ray findings are non-specific but may include:
  - a mid-to-lower zone predominance of patchy consolidation
  - pleural effusions in around 30%

Management

- treat with erythromycin/clarithromycin



Comparison of Legionella and Mycoplasma pneumonia



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Chest x-ray features of legionella pnuemonia are non-specific but includes a mid-to-lower zone predominance of patchy consolidation. Pleural effusions are seen in around 30%.

123

Next question

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Legionella pneumophila

Osmosis - YouTube 4 0

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A 37-year-old gentleman presents to the Emergency Department with acute onset shortness of breath and right sided pleuritic chest pain. He is known to be HIV positive and his latest CD4 count is 190/mm^3. The last clinic letter suggests he may be non-compliant with his anti-retroviral medication due to persistent diarrhoea. On examination, he has decreased breath sounds on the right with fine scattered crepitations in both lung fields. Oxygen saturations are 93% on breathing air. A chest x-Ray reveals a right sided pneumothorax measuring 1 cm at the hilum.

What pulmonary infection would you suspect?

- *Mycobacterium tuberculosis*
- *Pneumocystis jirovecii*
- *Aspergillus fumigatus*
- *Histoplasma capsulatum*
- *Haemophilus influenzae*

Submit answer

Reference ranges

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What pulmonary infection would you suspect?

Mycobacterium tuberculosis	2%
Pneumocystis jirovecii	92%
Aspergillus fumigatus	3%
Histoplasma capsulatum	3%
Haemophilus influenzae	1%

NICE guidance of pleural disease (pneumothorax):

- A spontaneous pneumothorax in someone known to be HIV-positive should warrant investigation for a diagnosis of *Pneumocystis jirovecii* pneumonia.

Invasive fungal infection (including *Pneumocystis jirovecii* pneumonia) is likely in this gentleman given the CD4 count <200/mm^3.

Discuss (1) | Improve

Next question >

HIV: *Pneumocystis jiroveci* pneumonia

Whilst the organism *Pneumocystis carinii* is now referred to as *Pneumocystis jiroveci*, the term *Pneumocystis carinii* pneumonia (PCP) is still in common use

- *Pneumocystis jiroveci* is an unicellular eukaryote, generally classified as a fungus but some authorities consider it a protozoa
- PCP is the most common opportunistic infection in AIDS

- all patients with a CD4 count < 200/mm^3 should receive PCP prophylaxis

Features

- dyspnoea
- dry cough
- fever
- very few chest signs

Pneumothorax is a common complication of PCP.

Extrapulmonary manifestations are rare (1-2% of cases), may cause

- hepatosplenomegaly
- lymphadenopathy
- choroid lesions

Investigation

- CXR: typically shows bilateral interstitial pulmonary infiltrates but can present with other x-ray findings e.g. lobar consolidation. May be normal
- exercise-induced desaturation
- sputum often fails to show PCP, bronchoalveolar lavage (BAL) often needed to demonstrate PCP (silver stain shows characteristic cysts)





Management

- co-trimoxazole
- IV pentamidine in severe cases
- aerosolized pentamidine is an alternative treatment for *Pneumocystis jiroveci* pneumonia but is less effective with a risk of pneumothorax
- steroids if hypoxic (if pO2 < 9.3kPa then steroids reduce risk of respiratory failure by 50% and death by a third)

AI + Q 123

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Pneumocystis pneumonia

Patient.info

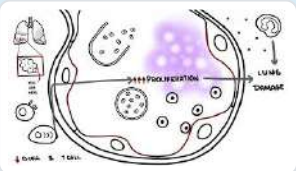
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Pneumocystis Jirovecii Pneumonia review

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Pneumocystis jirovecii - Pneumocystis pneumonia

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Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

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An 81-year-old nursing home resident presents with a 48-hour history of pyrexia, myalgia and coryza. On examination, her temperature is 37.9ºC and she is visibly coryzal. Her chest is clear to auscultation. Her chest X-ray is clear. Her relatives inform you that the nursing home has recently been closed to visitors due to an Influenza outbreak. Which of the following drugs should you also consider adding?

- Empirical antibiotics for pneumonia
- Zanamivir
- Oseltamivir
- Ribavirin
- Salbutamol nebulisers

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Reference ranges

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Empirical antibiotics for pneumonia	2%
Zanamivir	8%
Oseltamivir	88%
Ribavirin	1%
Salbutamol nebulisers	1%

Oseltamivir (Tamiflu) is the first-line treatment recommended for patients with suspected or confirmed Influenza A. Ribavirin has not be shown to be efficacious in the management of influenza. Zanamivir is useful in patients with a poor swallow or in those with suspected or confirmed exposure to oseltamivir-resistant influenza.

Her chest X-ray is clear and therefore there is no role for the addition of antibiotic therapy at this stage.

Salbutamol nebulisers can be useful in patients with wheeze, however, appropriate isolation precautions should be secured prior to the administration of nebulisers in patients with Influenza infection.

Discuss (3) | Improve

Next question >

Antiviral agents

Drug	Mechanism of action	Indications	Adverse effects/toxicity
Aciclovir	Guanosine analog, phosphorylated by	HSV, VZV	Crystalline nephropathy

Drug	Mechanism of action	Indications	Adverse effects/toxicity
	thymidine kinase which in turn inhibits the viral DNA polymerase		
Ganciclovir	Guanosine analog, phosphorylated by thymidine kinase which in turn inhibits the viral DNA polymerase	CMV	Myelosuppression/agranulocytosis
Ribavirin	Guanosine analog which inhibits inosine monophosphate (IMP) dehydrogenase, interferes with the capping of viral mRNA	Chronic hepatitis C, RSV	Haemolytic anaemia
Amantadine	Inhibits uncoating (M2 protein) of virus in cell. Also releases dopamine from nerve endings	Influenza, Parkinson's disease	Confusion, ataxia, slurred speech
Oseltamivir	Inhibits neuraminidase	Influenza	
Foscarnet	Pyrophosphate analog which inhibits viiral DNA polymerase	CMV, HSV if not responding to aciclovir	Nephrotoxicity, hypocalcaemia, hypomagnasaemia, seizures
Interferon-α	Human glycoproteins which inhibit synthesis of mRNA	Chronic hepatitis B & C, hairy cell leukaemia	Flu-like symptoms, anorexia, myelosuppression
Cidofovir	Acyclic nucleoside phosphonate, and is therefore independent of phosphorylation by viral enzymes (compare and contrast with aciclovir/ganciclovir)	CMV retinitis in HIV	Nephrotoxicity

Anti-retroviral agent used in HIV

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), didanosine, lamivudine, stavudine, zalcitabine

Protease inhibitors (PI)

- inhibits a protease needed to make the virus able to survive outside the cell
- examples: indinavir, nelfinavir, ritonavir, saquinavir

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz



Next question

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A 44 year-old lady presented to the medical outpatient clinic with leg swelling which had progressively increased over the last 2 years. Her past medical history consisted only of a hospital admission for malaria as a child. She had grown up in urban Nigeria and moved to the United Kingdom 6 years ago to work as a teacher, but had been forced to stop working due to mobility problems. She was a non-smoker and did not drink alcohol.

On examination, her temperature was 36.5ºC, heart rate 80 beats per minute, blood pressure 133/89 mmHg, respiratory rate 16 breaths per minute and oxygen saturations 98% on room air. Non-pitting leg swelling was apparent bilaterally extending proximally to the hips, with thickening of the overlying skin. Her chest was clear on auscultation and heart sounds were normal. The JVP was not elevated.

Investigations:

Haemoglobin	138 g/L
White cell count	7.0 * 10^9/l
Neutrophil count	4.8 * 10^9/l
Lymphocyte count	2.0 * 10^9/l
Eosinophil count	0.1* 10^9/l
Platelets	246 * 10^9/l

Sodium	142 mmol/L
Potassium	4.3 mmol/L
Urea	6.0 mmol/L
Creatinine	84 mol/L
Alkaline phosphatase	40 IU/L
Alanine aminotransferase	32 IU/L
Gamma-glutyl transferase	23 IU/L
Bilirubin	16 mol/L
Albumin	41 g/L
Fasting plasma glucose	5.3 mmol/L

What is the most likely causative organism?

- *Wuchereria bancrofti*

- *Brugia malayi*
- *Brugia timori*
- *Onchocerca volvulus*
- *Mansonella streptocerca*

Submit answer

Reference ranges

Score: **0%**

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A 44 year-old lady presented to the medical outpatient clinic with leg swelling which had progressively increased over the last 2 years. Her past medical history consisted only of a hospital admission for malaria as a child. She had grown up in urban Nigeria and moved to the United Kingdom 6 years ago to work as a teacher, but had been forced to stop working due to mobility problems. She was a non-smoker and did not drink alcohol.

On examination, her temperature was 36.5ºC, heart rate 80 beats per minute, blood pressure 133/89 mmHg, respiratory rate 16 breaths per minute and oxygen saturations 98% on room air. Non-pitting leg swelling was apparent bilaterally extending proximally to the hips, with thickening of the overlying skin. Her chest was clear on auscultation and heart sounds were normal. The JVP was not elevated.

Investigations:

Haemoglobin	138 g/L
White cell count	7.0 * 10^9/l
Neutrophil count	4.8 * 10^9/l
Lymphocyte count	2.0 * 10^9/l
Eosinophil count	0.1* 10^9/l
Platelets	246 * 10^9/l

Sodium	142 mmol/L
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Gamma-glutyl transferase	23 IU/L
Bilirubin	16 mol/L
Albumin	41 g/L
Fasting plasma glucose	5.3 mmol/L

What is the most likely causative organism?

Wuchereria bancrofti 85%

Brugia malayi	5%
Brugia timori	2%
Onchocerca volvulus	6%
Mansonella streptocerca	2%

This patient has presented with elephantiasis caused by chronic lymphatic inflammation and blockage in the lower limbs. The condition is caused by infiltration of the lymphatic system by filarial nematodes, which are transmitted by mosquitoes. *Wuchereria bancrofti*, *Brugia malayi* and *Brugia timori* are all potential causative organisms, but *Wuchereria bacrofti* is by far the most common, accounting for 90% of cases. *Onchocerca volvulus* causes skin disease and river blindness, while *Mansonella streptocerca* causes skin disease alone.

Discuss (8) | Improve

Next question >

Helminths

Nematodes (roundworms)

Worm	Notes	Treatment
Strongyloides stercoralis	Larvae are present in soil and gain access to the body by penetrating the skin Features include diarrhoea, abdominal pain, papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks, larva currens: pruritic, linear, urticarial rash, if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered	Ivermectin and -bendazoles are used
Enterobius vermicularis (pinworm)	Threadworm infestation is asymptomatic in around 90% of cases, possible features include perianal itching, particularly at night; girls may have vulval symptoms	-bendazoles

Worm	Notes	Treatment
	Diagnosis may be made by the applying sticky plastic tape to the perianal area and sending it to the laboratory for microscopy to see the eggs	
Ancylostoma duodenale, *Necator americanus* (hookworms)	Larvae penetrate skin of feet; gastrointestinal infection → anaemia Thin-shelled ova	-bendazoles
Loa loa	Transmission by deer fly and mango fly Causes red itchy swellings below the skin called 'Calabar swellings', may be observed when crossing conjunctivae	Diethylcarbamazine
Trichinella spiralis	Typically develops after eating raw pork Features include fever, periorbital oedema and myositis (larvae encyst in muscle)	-bendazoles
Onchocerca volvulus	Causes 'river blindness'. Spread by female blackflies Features include blindness, hyperpigmented skin and possible allergic reaction to microfilaria	Ivermectin r**IVER**blindness = **IVER**mectin
Wuchereria bancrofti	Transmission by female mosquito Causes blockage of lymphatics → elephantiasis	Diethylcarbamazine
Toxocara canis (dog roundworm)	Transmitted through ingestion of infective eggs. Features include visceral larva migrans and retinal granulomas VISCious dogs → blindness	Diethylcarbamazine
Ascaris lumbricoides (giant roundworm)	Eggs are visible in faeces May cause intestinal obstruction and occasional migrate to lung (Loffler's syndrome)	-bendazoles

Cestodes (tapeworms)

Worm	Notes	Treatment
Echinococcus granulosus	Transmission through ingestion of eggs in dog faeces. Definite host is dog, which ingests hydatid cysts from sheep, who act as an intermediate host. Often seen in farmers. Features include liver cysts and anaphylaxis if cyst ruptures (e.g. during surgical removal)	- bendazoles
Taenia solium	Often transmitted after eating undercooked pork. Causes cysticercosis and neurocysticercosis, mass lesions in the brain 'swiss cheese appearance'	- bendazoles

Trematodes (flukes)

Worm	Notes	Treatment
Schistosoma haematobium	Hosted by snails, which release cercariae that penetrate skin. Causes 'swimmer's itch' - frequency, haematuria. Risk factor for squamous cell bladder cancer	Praziquantel
Paragonimus westermani	Caused by undercooked crabmeat, results in secondary bacterial infection of lungs	Praziquantel
Clonorchis sinensis	Caused by undercooked fish Features include biliary tract inflammation. Known risk factor for cholangiocarcinoma	Praziquantel
Fasciola hepatica (the liver fluke)	May cause biliary obstruction	Triclabendazole

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Score: **32%**

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A 28-year-old male presents to the emergency department complaining of shortness of breath. For the last week he has felt generally unwell with a headache, malaise and lethargy, but felt breathless this afternoon hence his presentation. On systems review, he also admits to having some generalised abdominal pain throughout the day yesterday associated with diarrhoea. He is otherwise fit and well, takes no regular medications and does not smoke. He admits to drinking one or two beers every evening and occasionally more at the weekends. He is a primary school teacher and lives with his wife and two small children, none of whom have been unwell recently. On examination, he has a temperature of 38.9ºC, a heart rate of 105 beats/minute and a blood pressure of 105/70 mmHg. His respiratory rate is 26 breaths/minute, his oxygen saturations 92% breathing room air and on auscultation to his chest there are a few crepitations bibasally. He has some routine blood tests performed.

Hb	109 g/L
MCV	105 fL
Platelets	390 * 10^9/L
WBC	16.5 * 10^9/L
CRP	240 mg/L
Bilirubin	50 µmol/L
ALT	40 u/L
ALP	135 u/L

What is the most likely diagnosis?

- Alcoholic liver disease
- Gilbert's syndrome
- Mycoplasma pneumonia
- Hypothyroidism
- Acute cholecystitis

Submit answer

Reference ranges

Score: **0%**

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WBC	16.5 * 10^9/L
CRP	240 mg/L
Bilirubin	50 µmol/L
ALT	40 u/L
ALP	135 u/L

What is the most likely diagnosis?

Alcoholic liver disease	3%
Gilbert's syndrome	6%
Mycoplasma pneumonia	87%
Hypothyroidism	1%
Acute cholecystitis	4%

Mycoplasma pneumonia is one of the atypical pneumonias and classically occurs in clusters every few years. It normally produces a fairly mild picture with fairly normal clinical findings although the chest x-ray may have marked changes. It is associated with cold agglutinins in around 50% of cases which can result in a cold autoimmune haemolytic anaemia - as in this case, explaining the

slight bilirubin rise and the raised MCV which represents a reticulocytosis. Other associations with mycoplasma infection include erythema multiforme, encephalitis, arthralgia and diarrhoea. Alcoholic liver disease is possible given this patients alcohol intake, bilirubin and MCV, but would be unlikely given his young age and no other clinical findings to support this. Gilbert's syndrome is hyperbilirubinaemia in the context of transient illness, viral or otherwise, and could explain many of the findings in this case but would not explain the raised MCV.

Discuss (6) | Improve

Next question >

Mycoplasma pneumoniae

Mycoplasma pneumoniae is a cause of atypical pneumonia which often affects younger patients. It is associated with a number of characteristic complications such as erythema multiforme and cold autoimmune haemolytic anaemia. Epidemics of *Mycoplasma pneumoniae* classically occur every 4 years. It is important to recognise atypical pneumonia as it may not respond to penicillins or cephalosporins due to it lacking a peptidoglycan cell wall.

Features

- the disease typically has a prolonged and gradual onset
- flu-like symptoms classically precede a dry cough
- bilateral consolidation on x-ray
- complications may occur as below

Complications

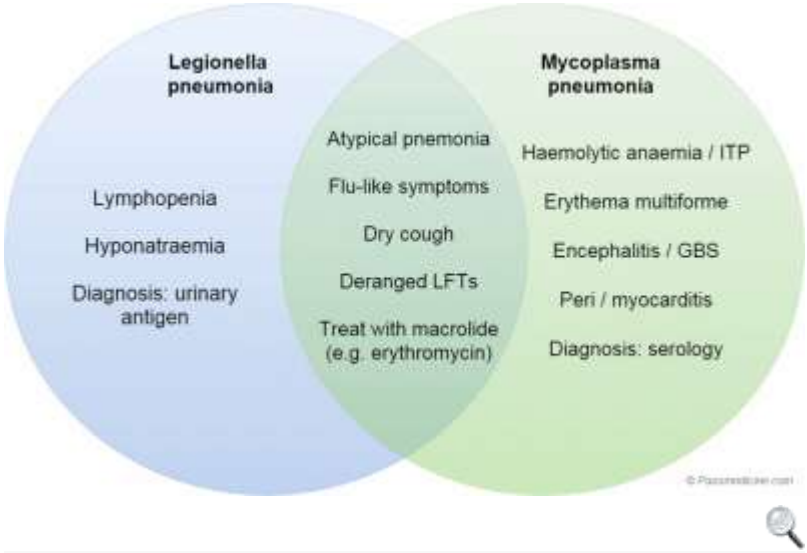
- cold agglutins (IgM): may cause an haemolytic anaemia, thrombocytopenia
- erythema multiforme, erythema nodosum
- meningoencephalitis, Guillain-Barre syndrome and other immune-mediated neurological diseases
- bullous myringitis: painful vesicles on the tympanic membrane
- pericarditis/myocarditis
- gastrointestinal: hepatitis, pancreatitis
- renal: acute glomerulonephritis

Investigations

- diagnosis is generally by Mycoplasma serology
- positive cold agglutination test → peripheral blood smear may show red blood cell agglutination

Management

- doxycycline or a macrolide (e.g. erythromycin/clarithromycin)



Comparison of Legionella and Mycoplasma pneumonia

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

11 6

Erythema multiforme

Suggest link

Report broken link

Media



Mycoplasma pneumoniae

Osmosis - YouTube 1 0

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A 39-year-old HIV-positive man complains of a two-week history of worsening headache, facial weakness and visual hallucinations. He also reports new onset eye pain. An MRI head reveals multiple ring-shaped contrast-enhancing lesions. He is regularly followed up in the HIV clinic and his latest CD4 count was 140 cells/mm^3. Which treatment should be started immediately?

- Valganciclovir
- Trimethoprim + sulfamethoxazole
- Dexamethasone
- Pyrimethamine + sulfadiazine
- Ethambutol + rifampicin

Submit answer

Reference ranges

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A 39-year-old HIV-positive man complains of a two-week history of worsening headache, facial weakness and visual hallucinations. He also reports new onset eye pain. An MRI head reveals multiple ring-shaped contrast-enhancing lesions. He is regularly followed up in the HIV clinic and his latest CD4 count was 140 cells/mm^3. Which treatment should be started immediately?

Valganciclovir	3%
Trimethoprim + sulfamethoxazole	9%
Dexamethasone	2%
Pyrimethamine + sulfadiazine	85%
Ethambutol + rifampicin	1%

The symptoms and MRI findings are highly suggestive of toxoplasmosis, a disease caused by the protozoan *Toxoplasma gondii*.

A 6-month treatment regimen of sulfadiazine and pyrimethamine should be commenced immediately. Patients should be given lifelong secondary prophylaxis.

Discuss (2) | Improve

Next question

Toxoplasmosis

Toxoplasma gondii is an obligate intracellular protozoan that infects the body via the gastrointestinal tract, lung or broken skin. It's oocysts release trophozoites which migrate widely around the body including to the eye, brain and muscle. The usual animal reservoir is the cat, although other animals such as rats carry the disease.

Immunocompetent patients

Most infections are asymptomatic. Symptomatic patients usually have a self-limiting infection, often having clinical features resembling infectious mononucleosis (fever, malaise, lymphadenopathy). Other less common manifestations include meningoencephalitis and

myocarditis.

Serology is the investigation of choice.

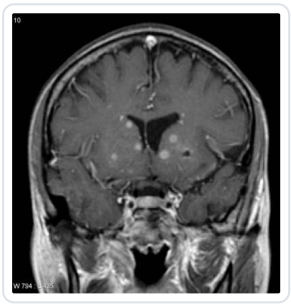
No treatment is usually required unless the patient has a severe infection or is immunosuppressed.

HIV/immunosuppressed patients

Cerebral toxoplasmosis accounts for around 50% of cerebral lesions in patients with HIV

- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring-enhancing lesions, mass effect may be seen
- management: pyrimethamine plus sulphadiazine for at least 6 weeks





Immunosuppressed patients may also develop a chorioretinitis secondary to toxoplasmosis.

Congenital toxoplasmosis

Congenital toxoplasmosis is due to transplacental spread from the mother. It causes a variety of effects to the unborn child including

- neurological damage
  - cerebral calcification
  - hydrocephalus
  - chorioretinitis
- ophthalmic damage
  - retinopathy
  - cataracts

123

Next question

Textbooks

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Extended textbook

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A 34-year-old man presents with back pain and leg weakness. This has been getting worse for the past two months and he now complains that he is 'bent over'. Over the past three months he describes feeling generally unwell, with a poor appetite and night sweats.

His past medical history includes hypothyroidism. He emigrated from Bangladesh around 6 months ago.

On examination he has a kyphosis. He is most tender over the lower part of the thoracic spine. Neurological examination of his lower limbs demonstrates reduced power in both legs.

A lumbar spinal film is shown below:



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What is the most likely diagnosis?

- Osteoporosis secondary to hypogonadism
- Pott's disease

- Actinomycosis
- Sarcoma
- Hodgkin's lymphoma

Submit answer

Reference ranges

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On examination he has a kyphosis. He is most tender over the lower part of the thoracic spine. Neurological examination of his lower limbs demonstrates reduced power in both legs.

A lumbar spinal film is shown below:



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What is the most likely diagnosis?

Osteoporosis secondary to hypogonadism	2%
Pott's disease	88%

Actinomycosis	6%
Sarcoma	1%
Hodgkin's lymphoma	2%

The x-ray shows destruction of T12 and L1, with a resultant gibbus deformity (a short-segment structural thoracolumbar kyphosis resulting in sharp angulation). The destructive process is centred on the T12/L1 disc space.

A MRI from the same patient is shown below:



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T12/L1 spondylodiscitis - with avid contrast enhancement involving the T11-L2 vertebral bodies, T12/L1 disc space and the adjacent paravertebral collections.

Hypogonadism is less likely given the systemic symptoms.

Discuss (1) Improve

Next question

Tuberculosis: types

Tuberculosis (TB) is an infection caused by *Mycobacterium tuberculosis* that most commonly affects the lungs. Understanding the pathophysiology of TB can be difficult - the key is to differentiate between primary and secondary disease.

Primary tuberculosis

A non-immune host who is exposed to *M. tuberculosis* may develop a primary infection of the lungs. A small lung lesion known as a Ghon focus develops. The Ghon focus is composed of tubercle-laden macrophages. The combination of a Ghon focus and hilar lymph nodes is known as a Ghon complex

In immunocompetent people, the initial lesion usually heals by fibrosis. Those who are immunocompromised may develop disseminated disease (miliary tuberculosis).

Secondary (post-primary) tuberculosis

If the host becomes immunocompromised the initial infection may become reactivated. Reactivation generally occurs in the apex of the lungs and may spread locally or to more distant sites. Possible causes of immunocompromise include:

- immunosuppressive drugs including steroids
- HIV
- malnutrition

The lungs remain the most common site for secondary tuberculosis. Extra-pulmonary infection may occur in the following areas:

- central nervous system (tuberculous meningitis - the most serious complication)
- vertebral bodies (Pott's disease)
- cervical lymph nodes (scrofuloderma)
- renal
- gastrointestinal tract





AI + Q 123

Next question

Textbooks

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Extended textbook

Links

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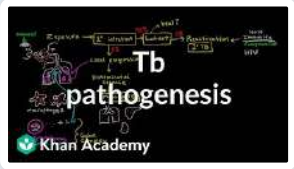
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2016 Tuberculosis guidelines

Suggest link

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Media



TB pathogenesis

Khan Academy - YouTube 1 0



Tuberculosis

Osmosis - YouTube 0 0

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A farmer presents with a tender lesion on his finger:



What is the most likely diagnosis?

- Paronychia
- Orf
- Tetanus
- Anthrax
- Hand, foot and mouth disease

Submit answer

Reference ranges

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A farmer presents with a tender lesion on his finger:



What is the most likely diagnosis?

Paronychia	15%
Orf	64%
Tetanus	1%
Anthrax	18%
Hand, foot and mouth disease	1%

The correct answer is **Orf**. Orf, also known as contagious ecthyma, is a zoonotic disease caused by a parapoxvirus that primarily affects sheep and goats. Humans can contract the disease through direct contact with infected animals or via fomites. The typical presentation includes a painful, red nodule on the hand or finger, often with central ulceration. This aligns with the farmer's occupation and the clinical picture presented.

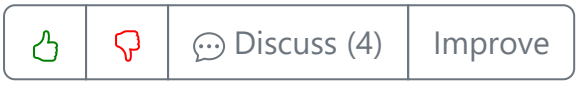
Paronychia is an infection of the skin around the nail, typically caused by staphylococci or streptococci bacteria. It presents with redness, swelling and tenderness at the side of the nail bed. Although this could be plausible given the patient's profession, it does not account for all his symptoms nor does it match the image provided.

Tetanus, caused by *Clostridium tetani*, is characterised by muscle stiffness and spasms due to neurotoxins produced by this bacterium following wound contamination. While farmers are at an

increased risk due to potential exposure to soil and animal faeces where *Clostridium tetani* spores reside, tetanus would not present as a single tender lesion on a finger.

Anthrax is another zoonotic disease caused by *Bacillus anthracis*. Cutaneous anthrax usually presents as a painless black eschar after an incubation period of 1-7 days post-exposure. However, in this case, there is no mention of exposure to livestock products such as hides or wool which would increase suspicion of anthrax.

Finally, **Hand, foot and mouth disease (HFMD)**, commonly caused by coxsackievirus A16 or enterovirus 71, primarily affects children under 10 years old. It presents with fever and vesicular lesions in the mouth and on hands/feet/buttocks. The age group affected and characteristic distribution of lesions make HFMD less likely in this scenario.



Next question

Orf

Orf is generally a condition found in sheep and goats although it can be transmitted to humans. It is caused by the parapox virus.

In animals

- 'scabby' lesions around the mouth and nose

In humans

- generally affects the hands and arms
- initially small, raised, red-blue papules
- later may increase in size to 2-3 cm and become flat-topped and haemorrhagic



Next question



Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

7 2

Orf

Suggest link

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A 26-year-old woman presents to the Emergency department some 2 weeks after returning from her honeymoon in the Seychelles. Since returning she has suffered from intermittent abdominal bloating and diarrhoea and feels she has lost a little weight. Physical examination reveals a blood pressure of 118/82 mmHg, and her pulse is 70 and regular. There is mild abdominal distension and her body mass index is 22 kg/m^2

Investigations:

Hb	110 g/l	Na^+	138 mmol/l
Platelets	210 * 10^9/l	K^+	4.0 mmol/l
WBC	11.2 * 10^9/l	Urea	6.9 mmol/l
Neuts	8.1 * 10^9/l	Creatinine	89 µmol/l
Lymphs	1.5 * 10^9/l	CRP	82 mg/l
Albumin	34 g/l		

Which of the following is the most appropriate intervention?

- Amoxicillin
- Ciprofloxacin
- Gluten free diet
- Lactose free diet
- Metronidazole

Submit answer

Reference ranges

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Investigations:

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Neuts	8.1 * 10^9/l	Creatinine	89 µmol/l
Lymphs	1.5 * 10^9/l	CRP	82 mg/l
Albumin	34 g/l		

Which of the following is the most appropriate intervention?

Amoxicillin	2%
Ciprofloxacin	21%
Gluten free diet	4%
Lactose free diet	12%
Metronidazole	61%

The most likely diagnosis, given the trip to a tropical region of Africa, intermittent bloating and diarrhoea, and an elevated CRP, is Giardiasis. Empirical treatment is often given, with either a course of metronidazole or a single dose of 2g tinidazole. Food intolerances may persist, particularly with respect to lactose, for some time after the initial infection.

Amoxicillin and ciprofloxacin would usually be used for listeriosis and campylobacter respectively, and neither infection is likely. Neither a wheat free diet nor a lactose-free diet is indicated given the likelihood of ongoing giardiasis, although many patients may suffer symptoms of lactose intolerance post-giardiasis.

Giardiasis

Giardiasis is caused by the flagellate protozoan *Giardia lamblia*. It is spread by the faeco-oral route.

Risk factors

- foreign travel
- swimming/drinking water from a river or lake
- male-male sexual contact

Features

- often asymptomatic
- non-bloody diarrhoea
    - steatorrhoea
- bloating, abdominal pain
- lethargy
- flatulence
- weight loss
- malabsorption and lactose intolerance can occur

Investigations

- stool microscopy for trophozoite and cysts: sensitivity of around 65%
- stool antigen detection assay: greater sensitivity and faster turn-around time than conventional stool microscopy methods
- PCR assays are also being developed

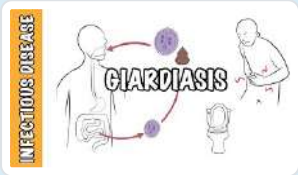
Treatment is with metronidazole.

Textbooks

High-yield textbook

Extended textbook

Media



Giardiasis

Armando Hasudungan - YouTube 2 1

Report broken media

Score: **32%**

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A 67-year-old woman is referred to the Emergency department by her GP because of an acute rise in serum creatinine. She has a history of Type 2 diabetes and takes lisinopril, amlodipine and indapamide as well as metformin and simvastatin, and has recently been prescribed trimethoprim for a UTI. Her blood pressure is 148/84 mmHg, with no postural drop on standing. There are no signs of fluid overload.

Na^+	140 mmol/l
K^+	4.9 mmol/l
Urea	5.8 mmol/l
Creatinine	162 µmol/l
Creatinine (one month earlier)	112 µmol/l

Which of the following is the most likely cause of her creatinine elevation?

- Amlodipine
- Lisinopril
- Post-renal failure
- Pre-renal failure
- Trimethoprim

Submit answer

Reference ranges

Score: **0%**

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Which of the following is the most likely cause of her creatinine elevation?

Amlodipine	0%
Lisinopril	12%
Post-renal failure	3%
Pre-renal failure	3%
Trimethoprim	82%

Trimethoprim is associated with a reversible elevation in serum creatinine because of a reduction in tubular excretion. Creatinine returns to normal once therapy ceases, and there is no effect on the GFR.

There is no suggestion of postural drop on blood pressure testing to support either pre-renal failure or amlodipine being the likely cause of creatinine elevation, and it's likely that the lisinopril has been prescribed for a prolonged period, rather than it being the cause of an acute rise in creatinine due to renal artery stenosis.

There are no symptoms of urinary obstruction to suggest post-renal failure.

Next question

Trimethoprim

Trimethoprim is an antibiotic, mainly used in the management of urinary tract infections.

Mechanism of action

- interferes with DNA synthesis by inhibiting dihydrofolate reductase
  - may, therefore, interact with methotrexate, which also inhibits dihydrofolate reductase

Adverse effects

- myelosuppression
- transient rise in creatinine: trimethoprim competitively inhibits the tubular secretion of creatinine resulting in a temporary increase which reverses upon stopping the drug
  - trimethoprim blocks the ENaC channel in the distal nephron, causing a hyperkalaemic distal RTA (type 4). It also inhibits creatinine secretion, often leading to an increase in creatinine by around 40 points (but not necessarily causing AKI)

Use in pregnancy

- the BNF advises that there is a: '*Teratogenic risk in first trimester (folate antagonist). Manufacturers advise avoid during pregnancy.*'

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **32%**

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A 36-year-old gentleman was admitted to the Medical Admission Unit. His principle complaint was watery diarrhoea which has been present for the last few weeks. He had not noticed the presence of blood or mucus in the stool. He also complained of transient pain on swallowing, and weight loss of 2 stones over the last year. He denied the presence of respiratory symptoms and abdominal pain. He had no past medical history of note from his GP records. He consumes 20 cans of standard strength lager per week, smokes 20 cigarettes per day and denies recreational drug use. He had no fixed abode.

On examination, he was unkempt and dishevelled, with a BMI of 17.1 kg/m^2. Cardiovascular and respiratory examinations were unremarkable except for an oxygen saturation of 94% on room air. His heart rate was 92/min, blood pressure 112/62 mmHg and temperature 36.7°C. Abdominal examination was also unremarkable though examination of the oral cavity revealed the presence of multiple aphthous ulcers. Examination of the neck revealed multiple small palpable cervical lymph nodes. Fundoscopy revealed the presence of white patches but otherwise, nil else and central and peripheral nervous system examination was otherwise normal. Skin examination revealed multiple pearly pink umbilicated nodules.

Initial investigations revealed the following:

Hb	122 g/l
Platelets	189 * 10^9/l
WBC	3.6 * 10^9/l

Chest x-ray: normal appearances of the heart and chest
ECG: 92bpm normal sinus rhythm, no other abnormalities seen
Urinalysis: normal
Stool MCS: normal interim results, pending further analysis

Which is the single investigation most likely to lead to the underlying diagnosis?

- CT head, chest, abdomen and pelvis
- Upper gastrointestinal endoscopy
- Bone marrow biopsy
- Colonoscopy
- HIV serology

Submit answer

Reference ranges

Score: **0%**

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Chest x-ray: normal appearances of the heart and chest
ECG: 92bpm normal sinus rhythm, no other abnormalities seen
Urinalysis: normal
Stool MCS: normal interim results, pending further analysis

Which is the single investigation most likely to lead to the underlying diagnosis?

CT head, chest, abdomen and pelvis	2%
Upper gastrointestinal endoscopy	5%
Bone marrow biopsy	2%
Colonoscopy	5%
HIV serology	85%

This gentleman has several conditions strongly suggestive of Acquired Immunodeficiency Syndrome (AIDS). He has features of cytomegalovirus (CMV) retinitis and oesophagitis caused by either CMV or other infections such as herpes simplex virus (HSV) and candidiasis. His loose stools are most likely representative of Cryptosporidium infection which would not be found on a conventional initial stool microscopy, culture and sensitivity test. He also has a skin infection in keeping with Molluscum contagiosum. Testing for HIV serology is, therefore, the best option.

Discuss (2) Improve

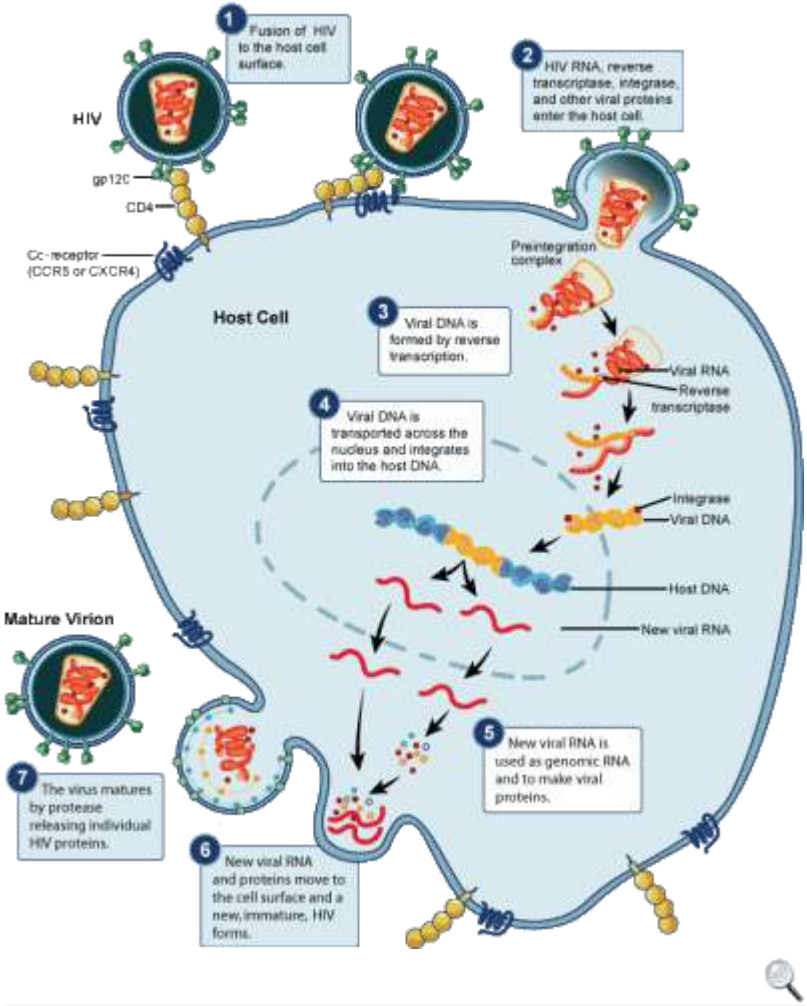
Next question

HIV: seroconversion

HIV seroconversion is symptomatic in 60-80% of patients and typically presents as a glandular fever-type illness. Increased symptomatic severity is associated with poorer long-term prognosis. It typically occurs 3-12 weeks after infection

Features

- sore throat
- lymphadenopathy
- malaise, myalgia, arthralgia
- diarrhoea
- maculopapular rash
- mouth ulcers
- rarely meningoencephalitis



An illustration model of the HIV Replication Cycle. Each step of the cycle is numbered and concisely described. Credit: NIAID

123

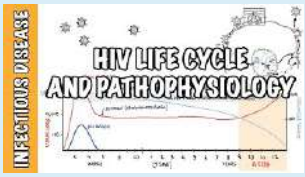
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Human immunodeficiency virus (HIV) - life cycle, pathophysiology, investigations, diagnosis

Armando Hasudungan - YouTube

2 0



HIV

Osmosis - YouTube

4 2

Report broken media

Score: **32%**

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A 38-year-old male presented to the Emergency Department complaining of feeling unwell. He complained of a headache that had been present for the last two days which he described a continuous ache worse in the morning, as well as a sore throat. He later developed diarrhoea, opening his bowels three times per day passing watery loose stool. Since then he has deteriorated, complaining of nausea and vomiting on the day of admission. He also noted a new rash on his chest which developed over the last few hours and complained of pains in his muscles. He had returned two weeks ago from a six-month backpacking expedition around Africa and Far East Asia; he was prescribed doxycycline as antimalarial prophylaxis but had discontinued them two weeks into his trip after suffering from severe sunburns. He had not taken recommended vaccinations prior to his trip as he was engaged with work commitments. His past medical history comprised of asthma and depression for which he was prescribed sertraline 100mg OD and Clenil modulite 2 puffs BD.

Examination revealed the presence of an unwell gentleman. His temperature was 40.1°C, heart rate 122 bpm, respiratory rate 22/min, oxygen saturations 95% on air and he had a blood pressure of 94/68 mmHg. He had a plethoric facial appearance, and multiple petechiae were noted on his chest and face. Examination of his cardiovascular system revealed the presence of normal heart sounds with a JVP of 2cm and a bounding radial pulse and vasodilated peripheries. Examination of his respiratory system revealed tachypnoea but with vesicular breath sounds. Examination of the gastrointestinal and neurological systems was unremarkable, with no neck stiffness, negative Kernig's and Brudzinski's signs. His GCS was 15. Multiple insect bites were noted on his legs and arms.

He was promptly transferred to the resuscitation area where two large bore cannulae were inserted and aggressive intravenous fluid resuscitation was commenced. A urinary catheter was inserted. The doctor inserting the cannulae noted that the sites of venepuncture continued to ooze despite the application of continual mechanical pressure.

Initial investigations revealed the following results:

Hb	102 g/l
Platelets	$8 * 10^9$/l
WBC	$3.2 * 10^9$/l
Neutrophils	$2.2 * 10^9$/l
Lymphocytes	$0.8 * 10^9$/l
Monocytes	$0.1 * 10^9$/l
Eosinophils	$0.1 * 10^9$/l
ESR	48 mm/hr

PTT	17 (NR 12-14s)
APTT	53 (NR 30-46s)
Fibrinogen	0.6 (NR 2-4g/l)
D-Dimer	428 (NR <230ng/ml)

Na^+	130 mmol/l
K^+	6.2 mmol/l
Urea	18.1 mmol/l
Creatinine	262 µmol/l
CRP	96 mg/l

Bilirubin	28 µmol/l
ALP	162 u/l
ALT	122 u/l
Total protein	52 g/l
Albumin	22 g/l

Blood MCS x3: awaiting result
Urine MCS: awaiting result
Thick film: negative
Thin film: awaiting result

Portable chest x-ray: normal appearance of heart and lung fields
ECG: sinus tachycardia 122bpm
Urinalysis: trace protein, ketones ++, nil else

What is the most likely diagnosis?

- ○ Viral haemorrhagic fever ×
- ○ Malaria ×
- ○ Typhus ×
- ○ Thrombotic thrombocytopaenic purpura ×
- ○ Typhoid fever ×

Submit answer

Reference ranges

Score: **0%**

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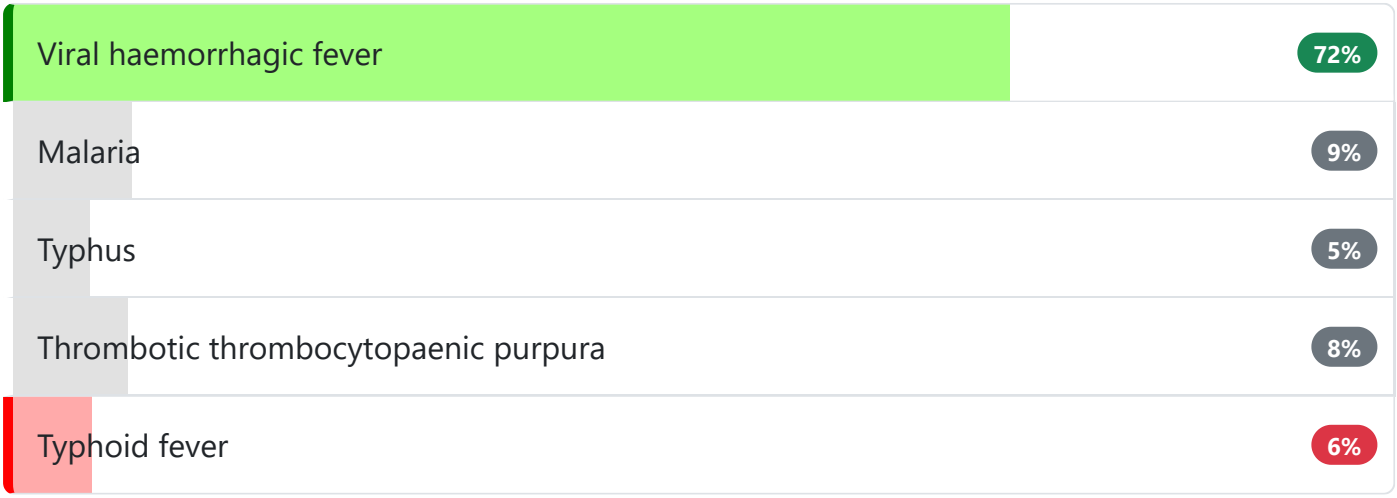
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Urinalysis: trace protein, ketones ++, nil else

What is the most likely diagnosis?



The Viral Haemorrhagic Fevers describe infection by a group of RNA viruses which include Yellow

fever, Lassa fever and Ebola virus. Given the current outbreak in West Africa, this group of infections must be considered as a differential in returning travellers presenting with a fever. They are characterised by initially non-specific symptoms such as fever, headache, vomiting, sore throat, diarrhoea and myalgic muscle pains, progressing to shock, renal failure and the presence of disseminated intravascular coagulation (DIC). Investigations characteristically reveal an anaemia with thrombocytopaenia, low lymphocyte count, deranged liver function and the presence of DIC. This condition requires intensive supportive care and early diagnosis is imperative. There are of course many differentials, malaria being a key one here, but the absence of parasites on thick film make this a less likely diagnosis.

Discuss (7) | Improve

Next question >

Viral haemorrhagic fevers

Viral haemorrhagic fevers (VHFs) are a group of viral infections that result in presentations ranging from a flu-like illness to multisystem failure.

Examples of viral haemorrhagic fever (VHF) include:

- Flaviviridae: dengue, yellow fever
- Arenaviridae: Lassa fever
- Filoviridae: Ebola virus, Marburg virus
- Bunyaviridae: Hantaviruses, Crimean-Congo haemorrhagic fever, Rift Valley fever

Features

- flu-like symptoms
- abdominal pain
- haemorrhage
  - petechiae, bruising
  - bloody diarrhoea, haematemesis, haemoptysis
  - disseminated intravascular coagulation
- multiorgan failure

Prevention

- a yellow fever vaccine is available
- a dengue and Ebola vaccine have been developed but are now widely used at the current time
- no vaccines are available for the other VHFs

Management

- treatment is generally supportive

- ribavirin is used to treat Lassa fever

The more common VHFs such as dengue, Ebola and yellow fever are covered in more detail elsewhere in the notes.

Lassa fever

Lassa fever is contracted by contact with the excreta of infected African rats (Mastomys rodent) or by person-to-person spread.

Marburg virus

Causes a VHF very similar to Ebola.

Risk factors for transmissions include caves/bats and primates.



Next question

Textbooks

High-yield textbook

Extended textbook

Links

HPA

2 1

Management of viral haemorrhagic fever

Suggest link Report broken link

Score: **32%**

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A 17-year-old man is referred to the infectious disease department with a sore throat, fever and lymphadenopathy. He has been generally unwell for the previous 3 days but presented as he noticed some yellowing of his eyes. His routine blood test show-

Bilirubin	55 µmol/l
ALP	54 u/l
ALT	402 u/l
AST	188 u/l
γGT	17 u/l
Albumin	43 g/l

He has no travel or sexual history and has never used intravenous drugs. What is the most likely causative organism?

- Viridans group Streptococci
- Hepatitis B virus
- *Echinococcus granulosus*
- Hepatitis C virus
- Epstein Barr virus (EBV)

Submit answer

Reference ranges

Score: **0%**

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γGT	17 u/l
Albumin	43 g/l

He has no travel or sexual history and has never used intravenous drugs. What is the most likely causative organism?

Viridans group Streptococci	4%
Hepatitis B virus	3%
Echinococcus granulosus	5%
Hepatitis C virus	2%
Epstein Barr virus (EBV)	85%

Epstein-Barr virus (EBV) is a herpes virus which commonly leads to infectious mononucleosis. This presents, often in teenagers and young adults with fevers, painful throat, lymphadenopathy and flu-like symptoms. Although it can last longer the symptoms are usually self-limiting within 2 weeks. Acute infection often leads to splenomegaly or hepatosplenomegaly and although a transient hepatitis is common it can lead to clinical jaundice.

Discuss (2) | Improve

Next question >

Infectious mononucleosis

Infectious mononucleosis (glandular fever) is caused by the Epstein-Barr virus (EBV, also known as human herpesvirus 4, HHV-4) in 90% of cases. Less frequent causes include cytomegalovirus and HHV-6. It is most common in adolescents and young adults.

The classic triad of sore throat, pyrexia and lymphadenopathy is seen in around 98% of patients:

- sore throat
- lymphadenopathy: may be present in the anterior and posterior triangles of the neck, in contrast to tonsillitis which typically only results in the upper anterior cervical chain being enlarged
- pyrexia

Other features include:

- malaise, anorexia, headache
- palatal petechiae
- splenomegaly - occurs in around 50% of patients and may rarely predispose to splenic rupture
- hepatitis, transient rise in ALT
- lymphocytosis: presence of 50% lymphocytes with at least 10% atypical lymphocytes
- haemolytic anaemia secondary to cold agglutins (IgM)
- a maculopapular, pruritic rash develops in around 99% of patients who take ampicillin/amoxicillin whilst they have infectious mononucleosis

Symptoms typically resolve after 2-4 weeks.

Diagnosis

- heterophil antibody test (Monospot test)
    - NICE guidelines suggest FBC and Monospot in the 2nd week of the illness to confirm a diagnosis of glandular fever.

Management is supportive and includes:

- rest during the early stages, drink plenty of fluid, avoid alcohol
- simple analgesia for any aches or pains
- consensus guidance in the UK is to avoid playing contact sports for 4 weeks after having glandular fever to reduce the risk of splenic rupture

There is an interesting correlation between EBV and socioeconomic groups. Lower socioeconomic groups have high rates of EBV seropositivity, having frequently acquired EBV in early childhood when the primary infection is often subclinical. However, higher socioeconomic groups show a higher incidence of infectious mononucleosis, as acquiring EBV in adolescence or early adulthood results in symptomatic disease.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries

9 7

Infectious mononucleosis

Patient.info

7 3

Infectious mononucleosis review

Suggest link

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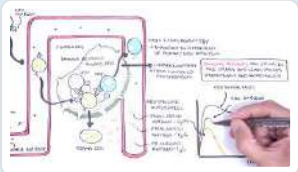
Media



Epstein Barr Virus (EBV) Diagnosis and Testing

macrophage - YouTube

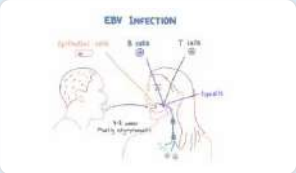
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Epstein Barr Virus and Infectious Mononucleosis (pathophysiology, investigations and treatment)

Armando Hasudungan - YouTube

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EBV and Mononucleosis - Pathogenesis and Clinical Presentation

macrophage - YouTube

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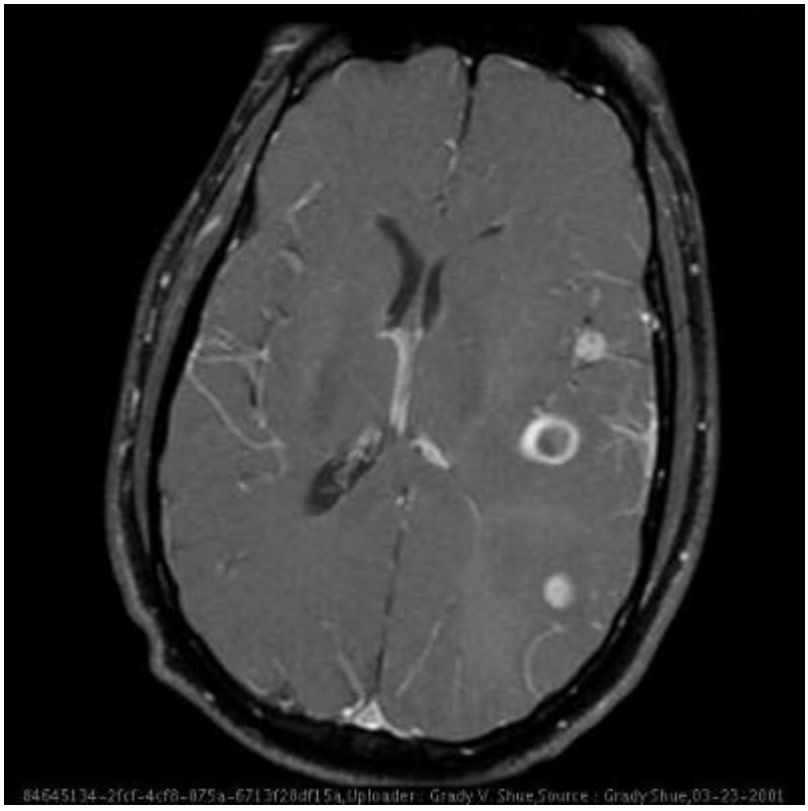
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A 50-year-old man with HIV presents to the emergency department with fits for 10 minutes. His vital signs are temperature 39 ºC, blood pressure 110/75 mmHg, and pulse 87 /minute. Examination reveals a Glasgow coma scale (GCS) score of 11 (E3V4M4). There is no focal neurological deficit.

Laboratory tests are requested as shown below:

Hb	135 g/L	(135-180)
Platelets	300 * 10^9/L	(150 - 400)
WBC	4.5 * 10^9/L	(4.0 - 11.0)
CD4 cell count	120 cells/mm^3	(500 - 1500)

An MRI brain is requested, as shown below:



© Image used on license from Radiopaedia

What is the most appropriate treatment for this patient?

- Intravenous amphotericin-B
- Methotrexate with whole brain irradiation

- Oral pyrimethamine-sulfadiazine
- Oral trimethoprim-sulfamethoxazole (TMP-SMX)
- Surgical excision

Submit answer

Reference ranges

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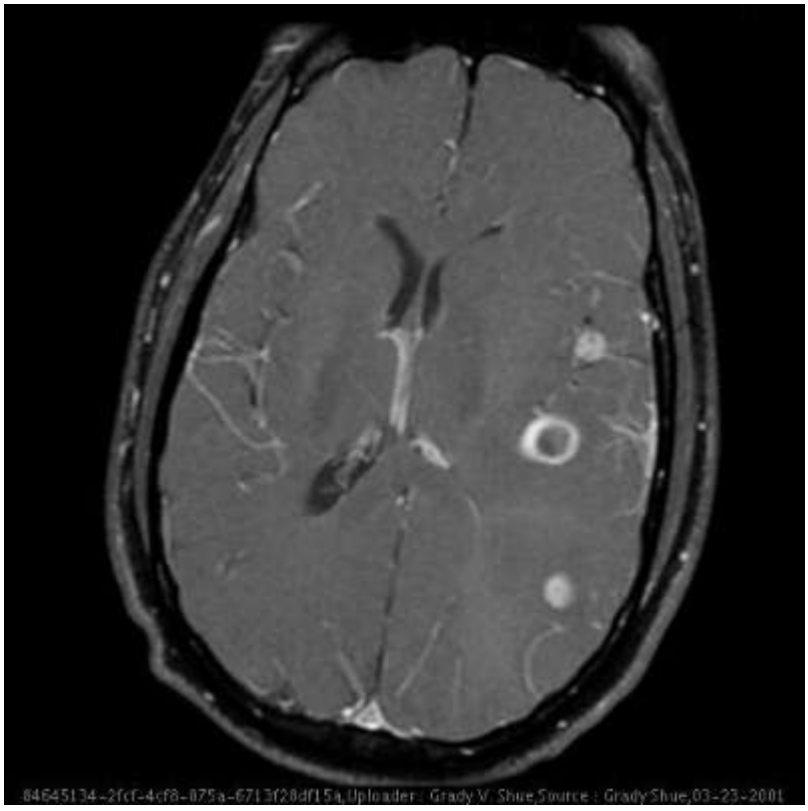
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A 50-year-old man with HIV presents to the emergency department with fits for 10 minutes. His vital signs are temperature 39 ºC, blood pressure 110/75 mmHg, and pulse 87 /minute. Examination reveals a Glasgow coma scale (GCS) score of 11 (E3V4M4). There is no focal neurological deficit.

Laboratory tests are requested as shown below:

Hb	135 g/L	(135-180)
Platelets	300 * 10^9/L	(150 - 400)
WBC	4.5 * 10^9/L	(4.0 - 11.0)
CD4 cell count	120 cells/mm^3	(500 - 1500)

An MRI brain is requested, as shown below:



© Image used on license from Radiopaedia

What is the most appropriate treatment for this patient?

Intravenous amphotericin-B	6%
Methotrexate with whole brain irradiation	2%

Oral pyrimethamine-sulfadiazine	83%
Oral trimethoprim-sulfamethoxazole (TMP-SMX)	8%
Surgical excision	1%

The most appropriate treatment option is **oral pyrimethamine-sulfadiazine** because the scenario describes a patient with HIV-related cerebral toxoplasmosis. MRI reveals intra-cerebral ring-enhancing lesions with peri-lesional oedema. History of fits in an HIV-positive patient along with the MRI findings in this scenario confirms the diagnosis of cerebral toxoplasmosis. A 3-month treatment with pyrimethamine-sulfadiazine is the most appropriate management option.

Cerebral toxoplasmosis is not treated with **intravenous amphotericin B** . Amphotericin B treats cryptococcal meningoencephalitis, which is another important neuro-complication of HIV. The MRI scan in cryptococcal meningitis shows cryptococcomas, meningeal enhancement, abscesses, and gelatinous pseudocysts.

Methotrexate with whole brain irradiation is the most appropriate treatment for primary CNS lymphoma which is sometimes indistinguishable from cerebral toxoplasmosis in HIV patients. In this scenario, an important differentiating feature of primary CNS lymphoma and cerebral toxoplasmosis is the presence of a ring-enhancing lesion on MRI. Primary CNS lymphoma is characterised by a single mass lesion on an MRI scan that enhances homogeneously. When there is diagnostic uncertainty, a 3-month treatment with pyrimethamine-sulfadiazine is given.

Prophylaxis with **oral trimethoprim-sulfamethoxazole (TMP-SMX)** is given against cerebral toxoplasmosis in HIV patients when the CD4 count is below 100 cells /mm^3. The patient in this scenario requires definitive treatment and not chemoprophylaxis with TMP-SMX because he has already developed cerebral toxoplasmosis, and has a CD4 count of above 100 /mm^3.

Low-grade primary CNS lymphoma in HIV can be treated by **surgical excision** . Surgical excision has no role in treating cerebral toxoplasmosis, which is the case here. Primary CNS lymphoma is the most common HIV-associated brain tumour. The treatment of choice for primary CNS lymphoma is high-dose methotrexate with radiotherapy. Surgical excision can be considered for low-grade tumours.

Discuss (1) Improve

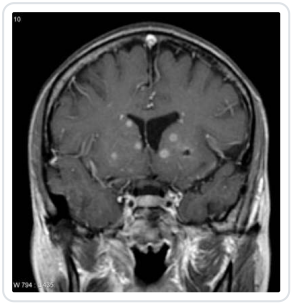
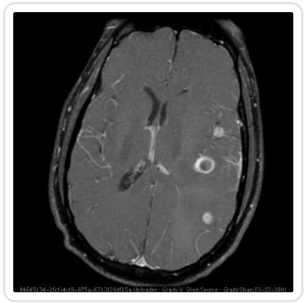
Next question ›

HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

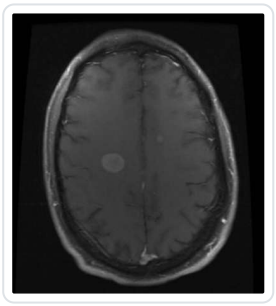
- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination
- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link

Report broken link

Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

0 1

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Score: **32%**

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A 23-year-old man presents for the second time to HIV clinic. He was found to have positive HIV serology on opportunistic screening in a local GUM clinic and was referred to HIV services. He had originally presented with dysuria and had been diagnosed with non-specific urethritis and treated with doxycycline. Following his diagnosis, he has separated with his partner, as she feared contracting HIV from him. He is anxious with managing his disease and is keen to start treatment. He has no other symptoms, feeling systemically well and no significant past medical history.

During his previous consultation, his diagnosis was discussed with him, and the role of antiretroviral treatment was fully explained.

Blood tests:

Hb	134 g/l
Platelets	378 * 10^9/l
WBC	7.2 * 10^9/l
Na^+	141 mmol/l
K^+	4.1 mmol/l
Urea	4.6 mmol/l
Creatinine	64 µmol/l
HIV viral load	223 copies/ml
CD4 count	914 cells/mm^3

What is the most appropriate management plan offer?

- Start antiretroviral therapy and PCP prophylaxis
- Start antiretroviral therapy
- Start treatment when CD4 count becomes <350
- Start treatment when CD4 count becomes <550
- Start treatment once HIV related symptoms develop

Submit answer

Reference ranges

Score: **0%**

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HIV viral load	223 copies/ml
CD4 count	914 cells/mm^3

What is the most appropriate management plan offer?

Start antiretroviral therapy and PCP prophylaxis	10%
Start antiretroviral therapy	87%
Start treatment when CD4 count becomes <350	2%
Start treatment when CD4 count becomes <550	1%
Start treatment once HIV related symptoms develop	1%

This is a patient with newly diagnosed HIV and should be offered HIV treatment regardless of CD4 count or viral load. PCP prophylaxis is only necessary when the CD4 count becomes much lower.

Source:
'BHIVA Guidelines for the Treatment of HIV-1-positive Adults with Antiretroviral Therapy 2015.' BHIVA. , Aug. 2016.

Discuss (1) | Improve

Next question >

HIV: *Pneumocystis jiroveci* pneumonia

Whilst the organism *Pneumocystis carinii* is now referred to as *Pneumocystis jiroveci*, the term *Pneumocystis carinii* pneumonia (PCP) is still in common use

- *Pneumocystis jiroveci* is an unicellular eukaryote, generally classified as a fungus but some authorities consider it a protozoa
- PCP is the most common opportunistic infection in AIDS
- all patients with a CD4 count < 200/mm^3 should receive PCP prophylaxis

Features

- dyspnoea
- dry cough
- fever
- very few chest signs

Pneumothorax is a common complication of PCP.

Extrapulmonary manifestations are rare (1-2% of cases), may cause

- hepatosplenomegaly
- lymphadenopathy
- choroid lesions

Investigation

- CXR: typically shows bilateral interstitial pulmonary infiltrates but can present with other x-ray findings e.g. lobar consolidation. May be normal
- exercise-induced desaturation
- sputum often fails to show PCP, bronchoalveolar lavage (BAL) often needed to demonstrate PCP (silver stain shows characteristic cysts)





Management

- co-trimoxazole
- IV pentamidine in severe cases
- aerosolized pentamidine is an alternative treatment for *Pneumocystis jiroveci* pneumonia but is less effective with a risk of pneumothorax
- steroids if hypoxic (if pO2 < 9.3kPa then steroids reduce risk of respiratory failure by 50% and death by a third)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF 7 8

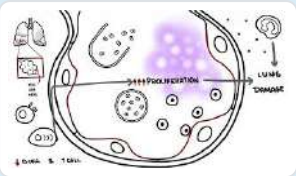
Pneumocystis pneumonia

Patient.info 6 6

Pneumocystis Jirovecii Pneumonia review

Suggest link Report broken link

Media



Pneumocystis jirovecii - Pneumocystis pneumonia

Armando Hasudungan - YouTube

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Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

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Score: **32%**

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A young 27 year-old man who has been travelling on a jungle expedition across south east Asia presents to the local doctors with a 3 day history of fever, headache and a widespread maculopapular rash. He has been kayaking through rivers, trekking through long grass and jungle, and he reports being bitten by mosquitos, flies and mites. On close inspection he has a black necrotic eschar on his leg. A malaria rapid diagnostic test (RDT) is negative.

What is the most appropriate management?

- Artemether / lumefantrine
- Benzylpenicilin
- Corticosteroids
- Doxycycline
- Supportive care

Submit answer

Reference ranges

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What is the most appropriate management?

Artemether / lumefantrine	3%
Benzylpenicilin	6%
Corticosteroids	1%
Doxycycline	85%
Supportive care	5%

The history describes a classical case of scrub typhus - and in particular the black eschar is the key diagnostic clue. Scrub typhus is caused by the tropical rickettsial bacteria *Orientia tsutsugamushi* and is transmitted by the bite of an infective trombiculoid 'chigger' mite. Its distribution is limited to South East Asia. Complications of disease include jaundice, meningoencephalitis, myocarditis, pneumonia and renal failure. However the disease can be easily treated with doxycyline.

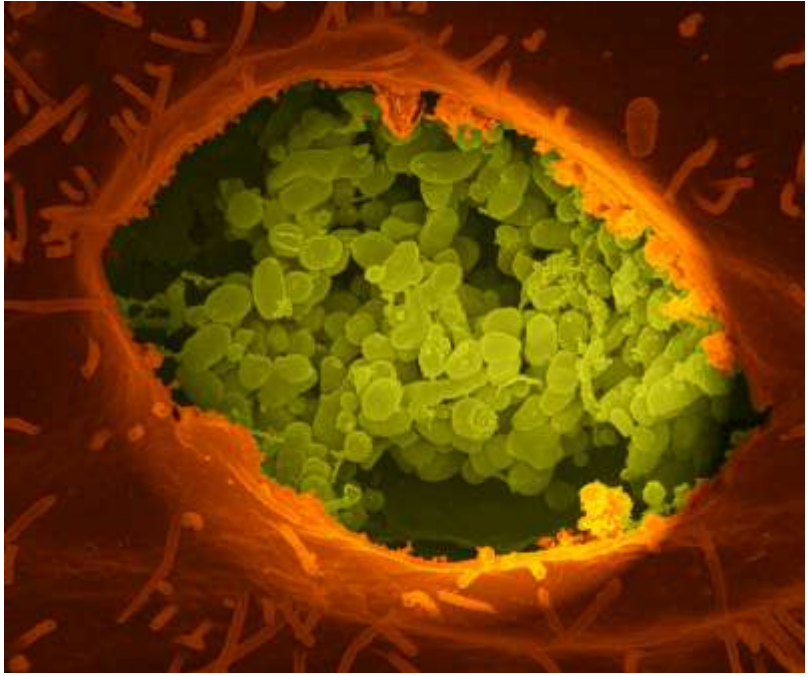
Similar disease presentations are seen in other continents. *Rickettsia ricketsii* causes the potentially fatal Rocky Mountain spotted fever (RMSF), but its distribution is limited to North and Central America. African tick typhus is a mild and self limiting infection with no reported deaths and caused by *R. africae*. Other tropical rickettsial disease include endemic murine typhus and epidemic louse borne typhus which are more serious forms of disease.

The other answers are trying to catch you out as fever, headache and rash are non specific symptoms. Artemether / lumefantrine is the correct treatment for uncomplicated *Plasmodium falciparum*; Benzylpenicillin is the treatment for Leptospirosis; Corticosteroid would be used to treat Katayama fever (acute schistosomiasis); and supportive care would be appropriate in the setting of uncomplicated dengue fever (typically an urban disease).

Rickettsiae ★

Rickettsiae are Gram-negative obligate intracellular parasites. Types of rickettsiae cause a variety of diseases that are typically characterised by fever, headache and rash. A notable exception is Q fever (cause by *Coxiella burnetti* which causes pneumonia but no rash. The Weil-Felix reaction is positive except in Q fever. Rickettsial diseases are all treated with tetracyclines.

Disease	Cause	Vector	Notes
Rocky Mountain spotted fever	*Rickettsia ricketsii*	Tick	Headache and fever are common Rash starts on the peripheries (wrist, ankles) before spreading centrally. It is initially maculopapular before becoming vasculitic Endemic to east coast of US
Q fever	*Coxiella burnetti*	No vector	No rash but causes pneumonia
Endemic typhus	*Rickettsia typhi*	Flea	Rash starts centrally then spreads to the peripheries
Epidemic typhus	*Rickettsia prowazekii*	Human body louse	
Ehrlichliosis	*Ehrlichia*	Tick	



A dry fracture of a Vero cell exposing the contents of a vacuole where *Coxiella burnetti* are busy growing. Note the intracellular nature of the organism. Credit: NIAID

123

Next question

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Media



Rickettsia rickettsii & other Rickettsia species

Osmosis - YouTube 5 0

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An 18-year-old male presents to the general medical take.

He presents with large grey lesions around his perineum and in his mouth which started 2 days ago. He reports that he has a more general rash on his trunk and the palms and soles of his feet. He generally feels unwell with malaise and fever. He has never had anything like this before but mentions that he did see a small ulcer on his scrotum a month ago that didn't hurt and healed on its own.

He reports that he is generally fit and well and has just moved to the area to start his university studies.

On examination, he has a symmetrical, widespread, macular papular rash on his trunk, limbs and the palms and soles of his feet. This is different from the large greyish lesions that you see in his mouth and perineum. He has general lymphadenopathy that is minimally tender on palpation. His chest is clear on auscultation, there are no added heart sounds and his abdomen is soft and non-tender.

Investigations show:

Haemoglobin	13g/dl
WCC	11 x 10^9/l
Platelets	352 x 10^9/l
CRP	89mg/L
Sodium	145 mmol/l
Potassium	3.8 mmol/l
Urea	4 mmol/l
Creatinine	82 µmol/l

What is the most likely diagnosis?

- Epstein Barr virus
- HIV
- Cytomegalovirus
- Herpes simplex virus

Treponema pallidum

Submit answer

Reference ranges

Score: **0%**

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CRP	89mg/L
Sodium	145 mmol/l
Potassium	3.8 mmol/l
Urea	4 mmol/l
Creatinine	82 µmol/l

What is the most likely diagnosis?

Epstein Barr virus	5%
HIV	5%
Cytomegalovirus	2%
Herpes simplex virus	6%

Treponema pallidum 82%

This young man presents with a symmetrical rash on his trunk, palms, and soles that is pathognomonic for secondary syphilis. He also has a further rash of grey lesions in the mouth and perineum associated with syphilis. Although he was not concerned about it, he has also mentioned that he had a painless genital ulcer one month ago that may have represented his primary syphilis infection. He has general lymphadenopathy and epitrochlear nodes are particularly suggestive of the diagnosis.

He is a young man that has recently started university and is likely to be enjoying this period of parties and new acquaintances. It would be worth exploring his sexual history further as syphilis is more common in men who have sex with men.

Patients are often co-infected with other sexually transmitted diseases and this patient should be further investigated for these including HIV.

Answers 1,2 and 3 may all be associated with lymphadenopathy, but one would not expect to get genital ulceration or grey mucosal lesions. HIV can be associated with a rash on the limbs, palms and soles.

Answer 4 is associated with genital ulceration, but they are usually multiple, extremely painful and do not cause the systemic symptoms described in this question.

https://www.bashh.org/documents/UK%20syphilis%20guidelines%202015.pdf

Discuss (3) Improve

Next question >

Syphilis

Syphilis is a sexually transmitted infection caused by the spirochaete *Treponema pallidum*. Infection is characterised by primary, secondary and tertiary stages. The incubation period is between 9-90 days

Primary features

- chancre - painless ulcer at the site of sexual contact
- local non-tender lymphadenopathy
- often not seen in women (the lesion may be on the cervix)

Secondary features - occurs 6-10 weeks after primary infection

- systemic symptoms: fevers, lymphadenopathy
- rash on trunk, palms and soles
- buccal 'snail track' ulcers (30%)
- condylomata lata (painless, warty lesions on the genitalia)





Tertiary features

- gummas (granulomatous lesions of the skin and bones)
- ascending aortic aneurysms
- general paralysis of the insane
- tabes dorsalis
- Argyll-Robertson pupil

Features of congenital syphilis

- blunted upper incisor teeth (Hutchinson's teeth), 'mulberry' molars
- rhagades (linear scars at the angle of the mouth)
- keratitis
- saber shins
- saddle nose
- deafness

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermIS.net 5 5

Picture of condylomata lata

Clinical Knowledge Summaries 8 4

Syphilis guidelines

DermNet NZ 12 5

Syphilis images

Suggest link Report broken link

Media



Syphilis

Osmosis - YouTube 5 0

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Score: **32%**

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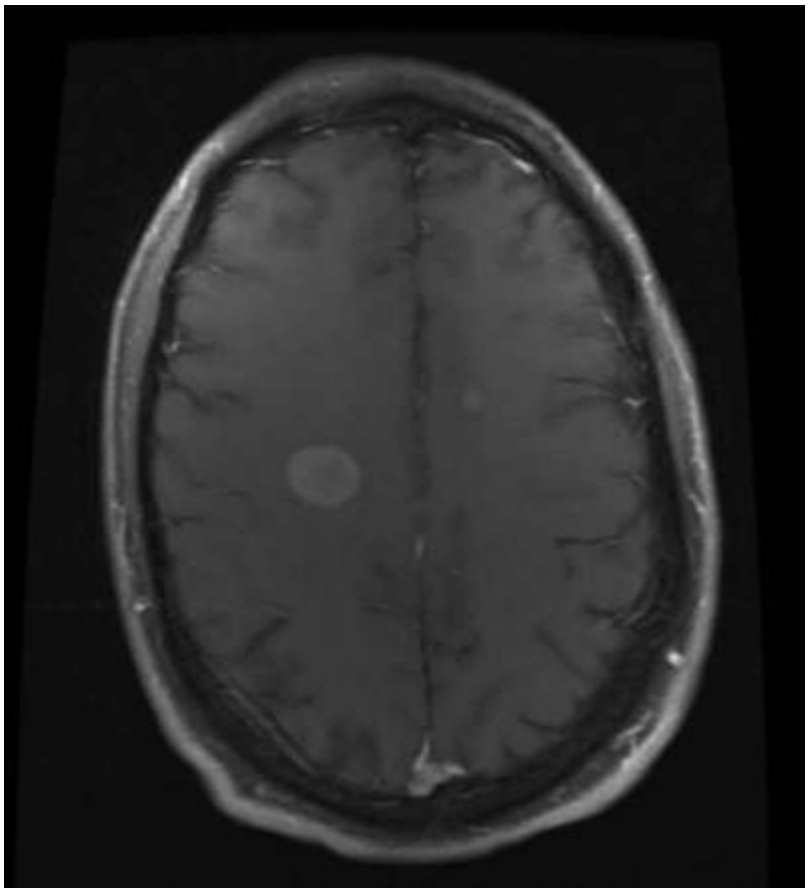
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A 45-year-old man presents to the emergency department with a 10-day history of progressively worsening headaches. Despite regular analgesia, the pain is not improving. He also complains of intermittent episodes of dizziness associated with nausea and vomiting. His past medical history includes human immunodeficiency virus for which he reports compliance with highly active antiretroviral therapy (HAART).

On examination, there is equal power and sensation in all 4 limbs. His cranial nerve and cerebellar examinations are unremarkable. Fundoscopic examination reveals bilateral papilloedema.

He is referred for neuroimaging, as shown below:



© Image used on license from Radiopaedia

What is the most likely diagnosis?

- CNS lymphoma
- Cryptococcal meningitis
- CMV encephalitis
- Progressive multifocal leukoencephalopathy

Toxoplasmosis

Submit answer

Reference ranges

Score: **0%**

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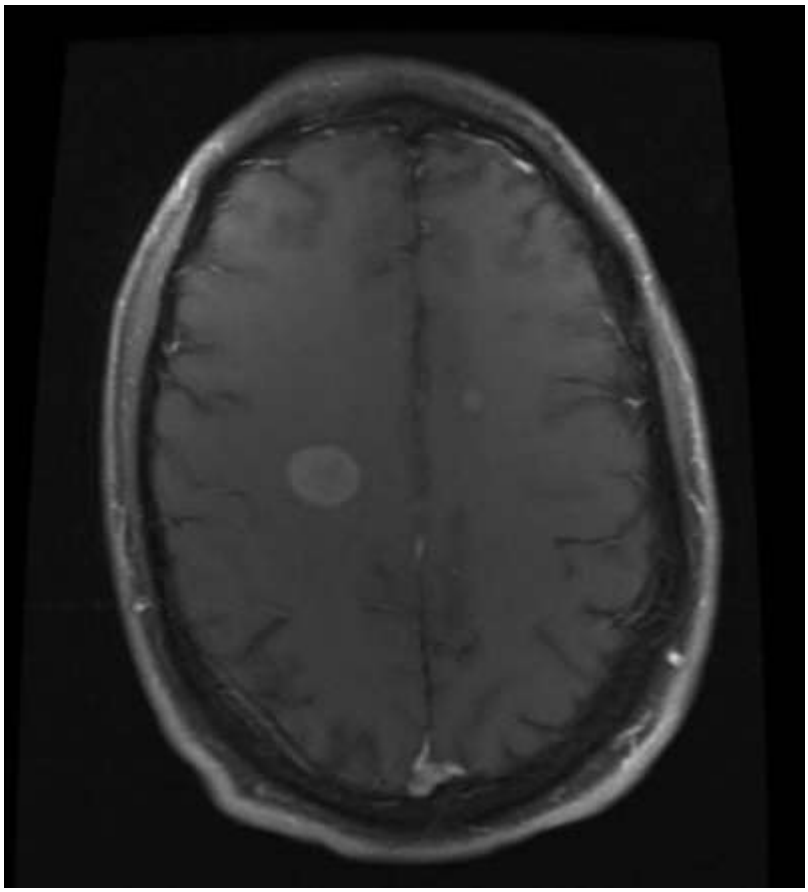
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He is referred for neuroimaging, as shown below:



© Image used on license from Radiopaedia

What is the most likely diagnosis?

CNS lymphoma	61%
Cryptococcal meningitis	4%
CMV encephalitis	1%
Progressive multifocal leukoencephalopathy	6%

Toxoplasmosis 28%

This patient's neuroimaging demonstrates 2 homogenous enhancing lesions; 1 larger lesion in the right hemisphere with 1 smaller lesion in the left hemisphere. Along with the history of worsening headaches associated with red-flag features (e.g. dizziness, vomiting), the presentation is in keeping with CNS lymphoma. CNS lymphoma accounts for up to 30% of cerebral lesions in patients with HIV and is associated with infection with the Ebstein-Barr virus. Like many cerebral lesions, presenting symptoms vary depending on the structures involved and include headache, nausea and vomiting, focal neurological signs and signs of raised intracranial pressure (e.g. papilloedema), as seen here.

Cryptococcus neoformans causes an opportunistic fungal infection that causes headache, fevers, confusion and signs of raised intracranial pressure. It is the most common fungal infection in patients with HIV. Imaging studies show leptomeningeal enhancement, cryptococcomas, hydrocephalus or dilated perivascular spaces. It does not account for lesions seen in this patient's MRI.

Cytomegalovirus (CMV) is a viral infection that commonly causes illness in patients with HIV, particularly in those with a CD4 count < 50 cells/mL. The most common form of CMV is CMV retinitis. However, CMV encephalitis is also common. On imaging, cerebral oedema may be seen. Meningoencephalitis and ventriculitis are also features of infection with CMV. These are not seen here, making CMV encephalitis unlikely.

Infection with JC virus is responsible for a condition known as progressive multifocal leukoencephalopathy, a demyelinating disease of the CNS. Symptoms include behavioural changes and alterations in speech, vision and movement. On imaging, single or multiple lesions may be seen, with demyelinating white matter lesions seen on MRI studies.

Toxoplasmosis is an opportunistic infection caused by the parasite *Toxoplasma gondii*. It is the most common cause of neurological brain abscesses in patients with HIV with symptoms including headache, fever and neurological changes including seizures, hemiplegia, cerebellar signs, or an acute confusional state. Domestic cats are a common source of transmission. Unlike CNS lymphoma, toxoplasmosis presents as ring-enhancing lesions on neuroimaging. These are not seen on this patient's scan, making it an unlikely diagnosis.

Discuss (4) Improve

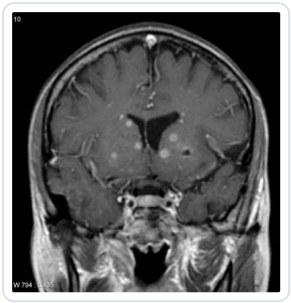
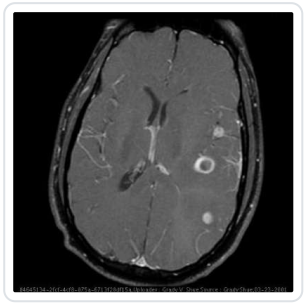
Next question ›

HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

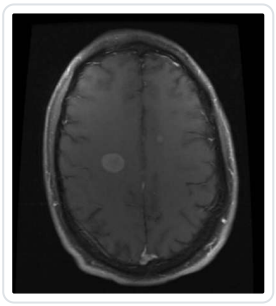
- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination
- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link

Report broken link

Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

0 1

Report broken media

Score: **32%**

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A 45-year-old man presents to the Emergency Department. He told the admitting doctor that he had fallen onto his left hip last night while he had been drinking at home. He had extensive bruising around his hip and some bony tenderness which resulted in a hip x-ray being taken.

His x-ray shows no fracture nor bony abnormality, but several 2x4mm specs of calcification are visible in his psoas and thigh muscles.

Later on, his wife asks to speak to you in confidence. She states that over the last year her husband's personality has gradually changed and he is sometimes forgetful. He lost his job 3 months ago as an engineer. He had worked for 8 years in Peru on an engineering project in his thirties. He drinks 8 units of alcohol per day and seems to have had a low mood for several years.

Yesterday he had fallen, lost consciousness, and had a jerking of his arms and legs lasting 1 minute. He had hit his hip on a coffee table and had wet himself. Afterwards, he felt tired and lethargic but had refused to go to hospital.

What is the most likely diagnosis?

- HIV dementia
- Alcohol withdrawal seizure
- Neurocysticercosis
- Emboli secondary to calcified aortic value
- Primary idiopathic epilepsy

Submit answer

Reference ranges

Score: **0%**

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Yesterday he had fallen, lost consciousness, and had a jerking of his arms and legs lasting 1 minute. He had hit his hip on a coffee table and had wet himself. Afterwards, he felt tired and lethargic but had refused to go to hospital.

What is the most likely diagnosis?

HIV dementia	9%
Alcohol withdrawal seizure	3%
Neurocysticercosis	86%
Emboli secondary to calcified aortic value	1%
Primary idiopathic epilepsy	1%

This is neurocysticercosis. It is the most common cause of epilepsy worldwide. Eggs of the Taenia Solium tapeworm are ingested, oncospheres hatch and migrate to peripheral sites (classically muscles and brain) and become cysticerci. They may remain asymptomatic or cause inflammatory pathology when the cysticerci die. They eventually become calcified.

Diagnosis is made with MRI or CT imaging. Calcified cysts in skeletal muscle may be found incidentally on x-ray. Serology may be helpful.

Management is with praziquantel or albendazole and prednisolone.

Discuss (4) | Improve

Next question

Helminths

Nematodes (roundworms)

Worm	Notes	Treatment
Strongyloides stercoralis	Larvae are present in soil and gain access to the body by penetrating the skin Features include diarrhoea, abdominal pain, papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks, larva currens: pruritic, linear, urticarial rash, if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered	Ivermectin and -bendazoles are used
Enterobius vermicularis (pinworm)	Threadworm infestation is asymptomatic in around 90% of cases, possible features include perianal itching, particularly at night; girls may have vulval symptoms Diagnosis may be made by the applying sticky plastic tape to the perianal area and sending it to the laboratory for microscopy to see the eggs	-bendazoles
Ancylostoma duodenale, *Necator americanus* (hookworms)	Larvae penetrate skin of feet; gastrointestinal infection → anaemia Thin-shelled ova	-bendazoles
Loa loa	Transmission by deer fly and mango fly Causes red itchy swellings below the skin called 'Calabar swellings', may be observed when crossing conjunctivae	Diethylcarbamazine
Trichinella spiralis	Typically develops after eating raw pork	-bendazoles

Worm	Notes	Treatment
	Features include fever, periorbital oedema and myositis (larvae encyst in muscle)	
Onchocerca volvulus	Causes 'river blindness'. Spread by female blackflies Features include blindness, hyperpigmented skin and possible allergic reaction to microfilaria	Ivermectin r**IVER**blindness = **IVER**mectin
Wuchereria bancrofti	Transmission by female mosquito Causes blockage of lymphatics → elephantiasis	Diethylcarbamazine
Toxocara canis (dog roundworm)	Transmitted through ingestion of infective eggs. Features include visceral larva migrans and retinal granulomas VISCious dogs → blindness	Diethylcarbamazine
Ascaris lumbricoides (giant roundworm)	Eggs are visible in faeces May cause intestinal obstruction and occasional migrate to lung (Loffler's syndrome)	-bendazoles

Cestodes (tapeworms)

Worm	Notes	Treatment
Echinococcus granulosus	Transmission through ingestion of eggs in dog faeces. Definite host is dog, which ingests hydatid cysts from sheep, who act as an intermediate host. Often seen in farmers. Features include liver cysts and anaphylaxis if cyst ruptures (e.g. during surgical removal)	-bendazoles
Taenia solium	Often transmitted after eating undercooked pork. Causes cysticercosis and neurocysticercosis, mass lesions in the brain 'swiss cheese appearance'	-bendazoles

Trematodes (flukes)

Worm	Notes	Treatment
Schistosoma haematobium	Hosted by snails, which release cercariae that penetrate skin. Causes 'swimmer's itch' - frequency, haematuria. Risk factor for squamous cell bladder cancer	Praziquantel
Paragonimus westermani	Caused by undercooked crabmeat, results in secondary bacterial infection of lungs	Praziquantel
Clonorchis sinensis	Caused by undercooked fish Features include biliary tract inflammation. Known risk factor for cholangiocarcinoma	Praziquantel
Fasciola hepatica (the liver fluke)	May cause biliary obstruction	Triclabendazole

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **32%**

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A 37-year-old man is referred to the medical outpatient clinic. He was born in Brazil but has been in the UK since university and now works in marketing.

He has been complaining of increasing breathlessness, worse on exertion, and is now breathless on climbing a flight of stairs. He has had several faints in the last few months, associated with feeling a 'funny' sensation in his chest, following which he blacks out. On one occasion he sustained a cut on his eye brow. There are no triggers to these episodes and there is a rapid recovery. He has never had any episodes of incontinence or tongue biting.

His past medical history is unremarkable. He was treated with several courses of antibiotics as a child in Brazil for an eye infection that persisted for several months. He has been seeing his GP for the past year for increasingly troublesome constipation. His medications are:

Senna 15mg at night
Movicol two sachets tds

On examination he has a displaced apex beat, normal heart sounds, and mild coarse crepitations at both lung bases.

What is the likely diagnosis?

- Chagas disease
- Endomyocardial fibrosis
- Churg Strauss Syndrome
- Hypertrophic Obstructive Cardiomyopathy
- Vasovagal syncope

Submit answer

Reference ranges

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Senna 15mg at night
Movicol two sachets tds

On examination he has a displaced apex beat, normal heart sounds, and mild coarse crepitations at both lung bases.

What is the likely diagnosis?

Chagas disease	91%
Endomyocardial fibrosis	4%
Churg Strauss Syndrome	2%
Hypertrophic Obstructive Cardiomyopathy	3%
Vasovagal syncope	0%

This is Chagas disease caused by *Trypanosoma cruzi*. Keys is the question are presence of Romana's sign (periorbital swelling following inoculation of Trypanosome) as a child, and heart failure and constipation in later life.

Trypanosomiasis

Two main form of this protozoal disease are recognised - African trypanosomiasis (sleeping sickness) and American trypanosomiasis (Chagas' disease).

Two forms of **African trypanosomiasis**, or **sleeping sickness**, are seen - *Trypanosoma gambiense* in West Africa and *Trypanosoma rhodesiense* in East Africa. Both types are spread by the tsetse fly. *Trypanosoma rhodesiense* tends to follow a more acute course. Clinical features include:

- Trypanosoma chancre - painless subcutaneous nodule at site of infection
- intermittent fever
- enlargement of posterior cervical lymph nodes
- later: central nervous system involvement e.g. somnolence, headaches, mood changes, meningoencephalitis

Management

- early disease: IV pentamidine or suramin
- later disease or central nervous system involvement: IV melarsoprol

American trypanosomiasis, or **Chagas' disease**, is caused by the protozoan *Trypanosoma cruzi*. The vast majority of patients (95%) are asymptomatic in the acute phase although a chagoma (an erythematous nodule at site of infection) and periorbital oedema are sometimes seen. Chronic Chagas' disease mainly affects the heart and gastrointestinal tract

- myocarditis may lead to dilated cardiomyopathy (with apical atophy) and arrhythmias
- gastrointestinal features includes megaoesophagus and megacolon causing dysphagia and constipation

Management

- treatment is most effective in the acute phase using azole or nitroderivatives such as benznidazole or nifurtimox
- chronic disease management involves treating the complications e.g., heart failure

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

8 3

Chaga's disease

Suggest link

Report broken link

Media



Sleeping Sickness - an introduction to African Trypanosomiasis

Let's Learn About Bugs - YouTube

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Chagas Disease - An overview

Let's Learn About Bugs - YouTube

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Chagas disease

Osmosis - YouTube

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Report broken media

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As the doctor on call you are called to review a 28-year-old male on the haematology ward. He recently underwent an allogenic stem cell transplant, which so far had been reasonably uncomplicated. Earlier in the week the patient had complained of coryzal symptoms and a dry cough. A nasopharyngeal aspirate (NPA) had been sent along with other routine tests 24 hours ago.

When you arrive to review the patient on the ward you note that he appears extremely sweaty. You take his temperature using a tympanic thermometer which reads 39.5°C. His heart rate is 95 beats per minute and his respiratory rate is 16 breaths per minute. Blood pressure is 122/76 mmHg. Physical examination is unremarkable and chest X-ray reveals clear lung fields.

You review the patients most recent results:

Hb	95 g/l	Na^+	134 mmol/l
Platelets	50 * 10^9/l	K^+	3.6 mmol/l
WBC	0.1 * 10^9/l	Urea	3.0 mmol/l
Neuts	0.1 * 10^9/l	Creatinine	65 µmol/l
Lymphs	0.0 * 10^9/l	CRP	12 mg/l
Eosin	0.0 * 10^9/l		
NPA	Influenza A		

What is the most appropriate treatment to commence?

- Amoxicillin
- Oseltamivir
- No treatment required
- Flucloxacillin
- Zanamivir

Submit answer

Reference ranges

Score: **0%**

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Lymphs	0.0 * 10^9/l	CRP	12 mg/l
Eosin	0.0 * 10^9/l		
NPA	Influenza A		

What is the most appropriate treatment to commence?

- Amoxicillin 2%
- Oseltamivir 74%
- No treatment required 6%
- Flucloxacillin 2%
- **Zanamivir** 17%

In this case they key points to realise are that the patient is immunosuppressed (neutropaenic post transplant) with symptoms and PCR confirmed Influenza A infection.

Whilst in most cases, oseltamivir (a neuraminidase inhibitor) is first line treatment for influenza, the BNF advises that for severely immunocompromised individuals zanamivir should be first line

treatment. This can help reduce the length of symptoms and prevent complications from developing during the course of the infection.

Discuss (7) | Improve

Next question >

Antiviral agents

Drug	Mechanism of action	Indications	Adverse effects/toxicity
Aciclovir	Guanosine analog, phosphorylated by thymidine kinase which in turn inhibits the viral DNA polymerase	HSV, VZV	Crystalline nephropathy
Ganciclovir	Guanosine analog, phosphorylated by thymidine kinase which in turn inhibits the viral DNA polymerase	CMV	Myelosuppression/agranulocytosis
Ribavirin	Guanosine analog which inhibits inosine monophosphate (IMP) dehydrogenase, interferes with the capping of viral mRNA	Chronic hepatitis C, RSV	Haemolytic anaemia
Amantadine	Inhibits uncoating (M2 protein) of virus in cell. Also releases dopamine from nerve endings	Influenza, Parkinson's disease	Confusion, ataxia, slurred speech
Oseltamivir	Inhibits neuraminidase	Influenza	
Foscarnet	Pyrophosphate analog which inhibits viiral DNA polymerase	CMV, HSV if not responding to aciclovir	Nephrotoxicity, hypocalcaemia, hypomagnasaemia, seizures
Interferon-α	Human glycoproteins which inhibit synthesis of mRNA	Chronic hepatitis B &	Flu-like symptoms, anorexia, myelosuppression

Drug	Mechanism of action	Indications	Adverse effects/toxicity
		C, hairy cell leukaemia	
Cidofovir	Acyclic nucleoside phosphonate, and is therefore independent of phosphorylation by viral enzymes (compare and contrast with aciclovir/ganciclovir)	CMV retinitis in HIV	Nephrotoxicity

Anti-retroviral agent used in HIV

Nucleoside analogue reverse transcriptase inhibitors (NRTI)

- examples: zidovudine (AZT), didanosine, lamivudine, stavudine, zalcitabine

Protease inhibitors (PI)

- inhibits a protease needed to make the virus able to survive outside the cell
- examples: indinavir, nelfinavir, ritonavir, saquinavir

Non-nucleoside reverse transcriptase inhibitors (NNRTI)

- examples: nevirapine, efavirenz



Next question

Textbooks

High-yield textbook

Extended textbook

Score: **32%**

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A 44-year-old man presents with a 1 day history of fever. He returned to the UK 12 days ago after visiting his family in northern Uganda. He has been resident in the UK for the last 20 years and returns home on average once a year. He does not routinely take malaria prophylaxis.

On examination his temperature is 39.4ºC, pulse is 86 beats per minute, his blood pressure is 115/78 mmHg and his oxygen saturations are 98% on air. The remainder of the physical examination is normal. A thick and thin blood film are sent to the lab which confirms *Plasmodium falciparum* malaria. The report reads 2.1% parasitaemia with the presence of schizonts.

What is the most appropriate initial management strategy?

- ○ Intravenous artesunate ×
- ○ Oral quinine sulfate ×
- ○ Oral chloroquine ×
- ○ Oral atovaquone-proguanil (Malarone) ×
- ○ Intravenous quinine ×

Submit answer

Reference ranges

Score: **32%**

1 ✓
2 ×
3 ✓
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What is the most appropriate initial management strategy?

Intravenous artesunate	84%
Oral quinine sulfate	1%
Oral chloroquine	4%
Oral atovaquone-proguanil (Malarone)	9%
Intravenous quinine	3%

This question examines your ability to differentiate severe malaria from uncomplicated malaria and then apply the correct therapy.

We are told the patient has been living in the UK for the last 20 years. As a result any natural immunity built up in earlier life would have dramatically reduced putting him at high risk of developing severe malaria with a relatively low level parasitaemia.

The most important information given is that schizonts are present in the blood film. Schizonts are the final step before red blood cells rupture and release more parasites (merozoites) into the blood.

Given his lack of immunity and an impending rise in parasitaemia it would be pertinent to consider this a case of severe malaria and treat accordingly with intravenous therapy early on. This excludes answers B, C and D. Furthermore chloroquine is only used routinely in the treatment of non falciparum malaria.

WHO guidelines recommend the use of intravenous artesunate for at least the first 24 hours until the patient can tolerate oral medication.

Discuss (4) Improve

Next question

Malaria: Falciparum

Falciparum malaria is the commonest, and most severe, type of malaria.

Clinical features

Falciparum malaria typically presents with symptoms suggestive of a flu-like illness. The classical triad of symptoms includes paroxysms of fever, chills, and sweating. These symptoms may occur every 48 hours corresponding to the erythrocytic cycle of the *Plasmodium falciparum* parasite. Fever is often high, intermittent, and may be accompanied by rigors. Initial manifestations can be non-specific and include malaise, headache, and myalgia, which might be mistaken for a viral syndrome.

General features of falciparum malaria

- fever: The hallmark of malaria, typically cyclical, often accompanied by sweating and sometimes rigors.
- gastrointestinal:
  - anorexia, nausea, vomiting, and abdominal pain are frequent
  - diarrhoea can occasionally be a feature, more commonly in children
  - patients may exhibit mild jaundice and occasional pruritus
- respiratory: Cough and, in some cases, mild tachypnoea may occur, though primary respiratory complications are rare in non-severe cases.
- musculoskeletal: Generalised body aches and joint pains are common.
- neurological:
  - headache is prominent and often severe.
  - dizziness and sleep disturbances may also be observed.
- cardiovascular: Tachycardia may be evident, and although hypotension is more typical of severe malaria, it can occasionally be seen in non-severe cases as well.
- haematological:
  - thrombocytopaenia is the most significant haematological finding and can occur in the absence of severe disease.
  - mild anaemia may also be present.
- renal: While acute kidney injury is associated with severe malaria, non-severe cases might show mild to moderate increases in creatinine or blood urea nitrogen levels.

Feature of severe falciparum malaria

- schizonts on a blood film
- parasitaemia > 2%
- hypoglycaemia

- acidosis
- temperature > 39 °C
- severe anaemia
- complications as below

Complications

- cerebral malaria: seizures, coma
- acute renal failure: blackwater fever, secondary to intravascular haemolysis, mechanism unknown
- acute respiratory distress syndrome (ARDS)
- hypoglycaemia
- disseminated intravascular coagulation (DIC)

Management

Uncomplicated falciparum malaria

- strains resistant to chloroquine are prevalent in certain areas of Asia and Africa
- the 2010 WHO guidelines recommend artemisinin-based combination therapies (ACTs) as first-line therapy
- examples include artemether plus lumefantrine, artesunate plus amodiaquine, artesunate plus mefloquine, artesunate plus sulfadoxine-pyrimethamine, dihydroartemisinin plus piperaquine

Severe falciparum malaria

- a parasite counts of more than 2% will usually need parenteral treatment irrespective of clinical state
- intravenous artesunate is now recommended by WHO in preference to intravenous quinine
- if parasite count > 10% then exchange transfusion should be considered
- shock may indicate coexistent bacterial septicaemia - malaria rarely causes haemodynamic collapse

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

WHO

9 7

Malaria treatment guidelines

Suggest link

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Media



Malaria

Osmosis - YouTube 5 1

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Score: **15.8%**

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On examination he has a temperature of 38.2 degrees, a heart rate of 98 beats per minute, a blood pressure of 110/70 and a respiratory rate of 20. He is lying calmly in the bed but is tender over the renal angle on the right side.

Investigations:

serum sodium	138 mmol/L (137-144)
serum potassium	5.5 mmol/L (3.5-4.9)
serum creatinine	240 mol/L (60-110)
haemoglobin	98 g/L (130-180)
white cell count	15.4 Ã— 109/L (4.0-11.0)
eosinophil count	0.89 Ã— 109/L (0.04-0.40)
platelet count	378 Ã— 109/L (150-400)
urine microscopy	red cells 2+, white cells 3+, protein 2+
ultrasound scan of abdomen	right sided hydronephrosis and hydroureter, fibrotic and calcified bladder

What is the most likely underlying diagnosis?

- *Schistosomiasis japonicum*
- Chronic vesico-ureteric reflux and secondary pyelonephritis
- Squamous cell carcinoma of the bladder
- *Schistosomiasis haematobium*
- *Schistosomiasis mansoni*

Submit answer

Reference ranges

Score: **32%**

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urine microscopy	red cells 2+, white cells 3+, protein 2+
ultrasound scan of abdomen	right sided hydronephrosis and hydroureter, fibrotic and calcified bladder

What is the most likely underlying diagnosis?

Schistosomiasis japonicum	3%
Chronic vesico-ureteric reflux and secondary pyelonephritis	3%
Squamous cell carcinoma of the bladder	1%
Schistosomiasis haematobium	80%
Schistosomiasis mansoni	12%

The clinical picture here is of an acute pyelonephritis with fever, rigours and tenderness over the renal angle. However blood analysis and imaging suggest there is another entity at play that has

predisposed the patient to this.

S. mansoni and *S japonicum*, *S. mekongi* and *S. intercalatum* produce eggs that can invade the bowel wall causing an intense inflammatory reaction that gives rise to loose bloody stools. Eggs can also migrate to liver through the portal venous system where they can elicit a granulomatous fibrosing reaction. This can eventually lead to the blockade of venous blood and flow. Portal venous hypertension results which can give rise to varicies and upper gastrointestinal bleeding.

S. haematobium on the other hand leads to granulomatous inflammation, ulceration of the vesicle and ureteral walls. Subsequent fibrosis can cause bladder neck obstruction, hydroureter and hydronephrosis. These changes can cause a chronic renal impairment and predispose to secondary bacterial infection as well as squamous cell carcinoma.

In addition, all schistosome species can result in immune complex deposition in the kidneys leading to a proteinuria and nephrotic syndrome.

Discuss (1) Improve

Next question

Schistosomiasis

Schistosomiasis, or bilharzia, is a parasitic flatworm infection. The three main species of schistosome are *S. mansoni*, *S. japonicum* and *S. haematobium*.

Acute infections

Acute symptoms typically only develop in people who travel to endemic areas, as they don't have any immunity to the worms.

Acute manifestations may include:

- swimmers' itch
- acute schistosomiasis syndrome (Katayama fever)
  - fever
  - urticaria/angioedema
  - arthralgia/myalgia
  - cough
  - diarrhoea
  - eosinophilia

Chronic infections

Schistosoma haematobium

These worms deposit egg clusters (pseudopapillomas) in the bladder, causing inflammation. The calcification seen on x-ray is actually calcification of the egg clusters, not the bladder itself.

Depending on the site of these pseudopapillomas in the bladder, they can cause an obstructive uropathy and kidney damage.

This typically presents as a 'swimmer's itch' in patients who have recently returned from Africa. Schistosoma haematobium is a risk factor for squamous cell bladder cancer.

Features

- frequency
- haematuria
- bladder calcification

Investigation

- for asymptomatic patients serum schistosome antibodies are generally preferred
- for symptomatic patients the gold standard for diagnosis is urine or stool microscopy looking for eggs

Management

- single oral dose of praziquantel

Schistosoma mansoni and *Schistosoma japonicum*

These worms mature in the liver and then travel through the portal system to inhabit the distal colon. Their presence in the portal system can lead to progressive hepatomegaly and splenomegaly due to portal vein congestion.

These species can also lead to complications of liver cirrhosis, variceal disease and cor pulmonale.

Schistosoma intercalatum and *Schistosoma mekongi*

These are less prevalent than the other three forms, but are both attributed to intestinal schistosomiasis.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians

2011 Schistosomiasis review

9 5

Suggest link

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Media



Schistosomiasis (Bilharzia)â€“ an overview

Let's Learn About Bugs - YouTube 6 1

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Score: **15.8%**

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A 42-year-old male from Bolivia presents with a 8 month history of progressive fatigue, dyspnoea and intermittent chest pains. He is a chronic smoker and during a recent severe episode of breathlessness had consulted his GP who prescribed amoxicillin and prednisolone to combine with his regular inhaler therapy. Shortly after this he experienced a febrile episode which had lasted 7 days before resolving. His breathlessness also worsened during this period.

On examination he has a temperature of 37.2 degrees, a heart rate of 98 beats per minute, a blood pressure of 110/70 mmHg and a respiratory rate of 24/min. His jugular venous pressure was raised, there was lower limb oedema to the mid shin and a pan systolic murmur heard best in inspiration in the left parasternal region. An ECG showed a prolonged PR interval.

What is the most likely underlying diagnosis?

- ○ Ischaemic heart disease ×
- ○ Chronic pulmonary emboli ×
- ○ Chronic Chagas cardiomyopathy ×
- ○ Sarcoidosis ×
- ○ HIV ×

Submit answer

Reference ranges

Score: **32%**

1 ✓
2 ×
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What is the most likely underlying diagnosis?

Ischaemic heart disease	1%
Chronic pulmonary emboli	2%
Chronic Chagas cardiomyopathy	92%
Sarcoidosis	2%
HIV	3%

Teasing out the infective aetiology in this question is the key to finding the answer. Chagas disease can present as an acute illness or as a chronic disease years after infection and is caused by the parasite *Trypanosoma cruzi*.

Acute disease is usually asymptomatic but may manifest as a febrile episode in association with a swelling over the site of inoculation (if in the skin this is called a chagoma if through the conjunctiva of the eye this is known as Romanas sign). In severe cases an acute myocarditis can evolve with a secondary pericardial effusion.

Chronic disease begins to develop weeks or months after the initial inoculation and in some cases does not present until years after the infection (as in this case). Chronic disease can effect the heart causing a biventricular cardiomyopthy with predominant right sided features as well as varying degrees of heart block and conductive abnormalities due to fibrous inflammation around amastigotes embedded in the heart muscle. Chest pain can also be common and does not imply an ischaemic component. Chronic disease can also effect the bowel causing dilation.

The first clue in the case is that the patient is from an area where disease is endemic. Secondarily

there is an absence of ischaemic related risk factors other than his smoking history and he is of a relatively young age. Perhaps the biggest clue of an infective cause is the febrile episode following steroids therapy. This likely represents a relapse of acute infection which is common with immunosupression. At this point you may be able to see trypomastigotes in the blood but they soon disappear on resolution of the fever.

Chronic PE is also a possibility given the predominance of right sided signs but does not explain the first degree heart block or fever following steroid therapy.

Discuss (4) | Improve

Next question >

Trypanosomiasis

Two main form of this protozoal disease are recognised - African trypanosomiasis (sleeping sickness) and American trypanosomiasis (Chagas' disease).

Two forms of **African trypanosomiasis**, or **sleeping sickness**, are seen - *Trypanosoma gambiense* in West Africa and *Trypanosoma rhodesiense* in East Africa. Both types are spread by the tsetse fly. *Trypanosoma rhodesiense* tends to follow a more acute course. Clinical features include:

- Trypanosoma chancre - painless subcutaneous nodule at site of infection
- intermittent fever
- enlargement of posterior cervical lymph nodes
- later: central nervous system involvement e.g. somnolence, headaches, mood changes, meningoencephalitis

Management

- early disease: IV pentamidine or suramin
- later disease or central nervous system involvement: IV melarsoprol

American trypanosomiasis, or **Chagas' disease**, is caused by the protozoan *Trypanosoma cruzi*. The vast majority of patients (95%) are asymptomatic in the acute phase although a chagoma (an erythematous nodule at site of infection) and periorbital oedema are sometimes seen. Chronic Chagas' disease mainly affects the heart and gastrointestinal tract

- myocarditis may lead to dilated cardiomyopathy (with apical atophy) and arrhythmias
- gastrointestinal features includes megaoesophagus and megacolon causing dysphagia and constipation

Management

- treatment is most effective in the acute phase using azole or nitroderivatives such as benznidazole or nifurtimox
- chronic disease management involves treating the complications e.g., heart failure

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

8 3

Chaga's disease

Suggest link Report broken link

Media



Sleeping Sickness - an introduction to African Trypanosomiasis

Let's Learn About Bugs - YouTube 7 0



Chagas Disease - An overview

Let's Learn About Bugs - YouTube 3 1



Chagas disease

Osmosis - YouTube 2 1

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Score: **15.8%**

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A 50-year-old man presents to the Emergency Department after becoming increasingly unwell over the previous two weeks. His initial symptoms were fevers increasing shortness of breath. In the last 24 hours he had experienced several episodes of haemoptysis and bloody diarrhoea precipitating his attendance to hospital.
Past medical history included a diagnosis of systemic lupus erythematous five years previously. This disease had been poorly controlled in recent months requiring the patient to take prednisolone 40 mg daily and hydroxychloroquine 400 mg daily. The patient was originally from Mali and had emigrated to the UK twenty years previously. He had returned to visit family in Africa earlier in the year and had not taken malaria prophylaxis whilst abroad.

Initial assessment revealed the patient to be very unwell. Respiratory rate was 32 with O2 saturations of 94 % on high flow oxygen. Blood pressure was 80/50 mmHg with no improvement in hypotension following aggressive fluid resuscitation. The patient was intubated and ventilated and transferred to the Intensive Care Unit for inotropic support.

Results of immediate investigations are listed below.

Chest x-ray: widespread diffuse pulmonary infiltrates; left sided pleural effusion; no free air under the diaphragm

Abdominal x-ray: multiple loops of distended small bowel; no evidence of free air

Hb	11.2 g/dl
Mean cell volume	83 fL
Platelets	150 * 10^9/l
WBC	18.7 * 10^9/l
Neutrophils	14.8 * 10^9/l
Lymphocytes	2.0 * 10^9/l
Monocytes	1.0 * 10^9/l
Eosinophils	0.9 * 10^9/l
Blood film	no abnormality detected
Internationalised normalised ratio	1.2

Na^+	124 mmol/l
K^+	4.9 mmol/l
Urea	15 mmol/l
Creatinine	190 µmol/l

C-reactive protein	284 mg/dL

Blood cultures: Gram negative cocci (positive after 24 hours)

Pleural fluid microscopy: filariform larvae

What is the most likely underlying diagnosis?

- ○ Caecal carcinoma ×
- ○ Strongyloides hyperinfection syndrome ×
- ○ Churg-Strauss vasculitis ×
- ○ Plasmodium falciparum infection ×
- ○ Disseminated haematogenous tuberculosis ×

Submit answer

Reference ranges

Score: **32%**

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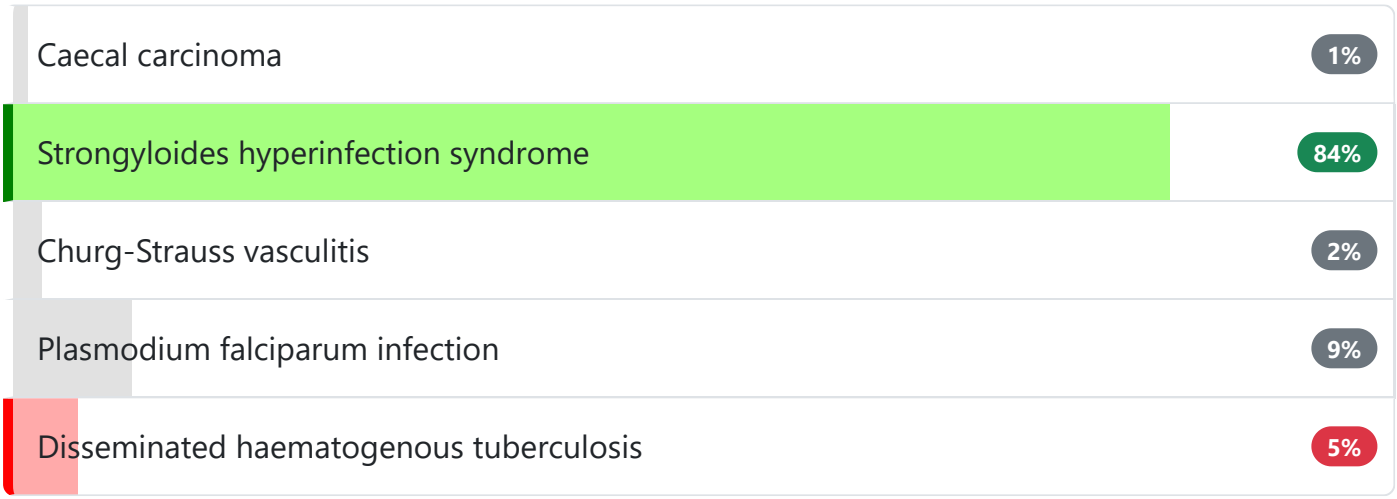
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Creatinine	190 µmol/l

C-reactive protein	284 mg/dL

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Pleural fluid microscopy: filariform larvae

What is the most likely underlying diagnosis?



Strongyloides hyperinfection syndrome occurs in immunosuppressed individuals with coexistent Strongyloides stercoralis infection. Uncontrolled proliferation of larvae occurs with dissemination to end organs.

As in this individual, systemic sepsis often occurs due to involvement of the gut wall allowing translocation of gut bacteria. Other features include pulmonary infiltrates, paralytic ileus, gastrointestinal bleeding and syndrome of inappropriate ADH secretion.

Filariform larvae can be found in body fluids by microscopy and it is this that confirms the diagnosis in this case. Due to immunosuppression, eosinophilia is often absent. Given the patient's country of origin, strongyloidosis is most likely to have been chronic, but could have been acquired on his recent trip to Africa.

Plasmodium falciparum infection can cause a similar presentation, although the normal blood film in this case makes this unlikely. Severe tuberculosis infection tends to predominantly involve the lungs with a varying degree of involvement of other organ systems.

Churg-Strauss vasculitis can cause severe abdominal symptoms including infarction and perforation. However, the normal eosinophil count, short history and immunosuppresion do not support this diagnosis.

Colonic carcinoma can lead to translocation of gut bacteria and gram-negative sepsis. However, this diagnosis would not explain all of the findings in this case and is not supported by the normal mean cell volume.

Strongyloides stercoralis

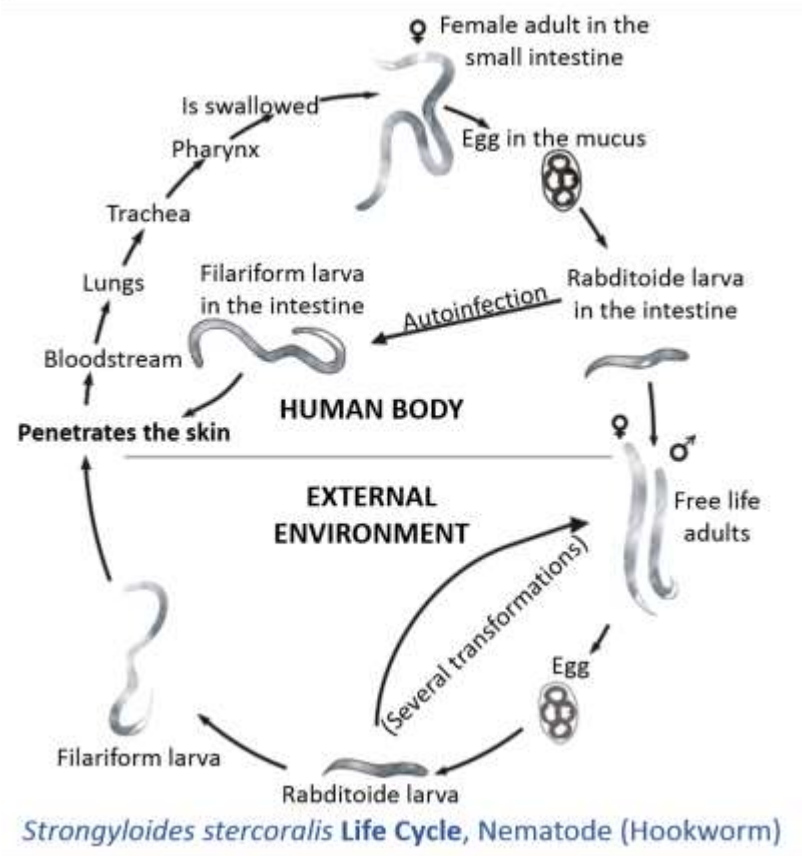
Strongyloides stercoralis is a human parasitic nematode worm. The larvae are present in soil and gain access to the body by penetrating the skin. Infection with *Strongyloides stercoralis* causes strongyloidiasis.

Features

- diarrhoea
- abdominal pain/bloating
- papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks
- larva currens: pruritic, linear, urticarial rash
- if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered

Treatment

- ivermectin and albendazole are used



Strongyloides stercoralis **Life Cycle**, Nematode (Hookworm)

Image sourced from Wikipedia

Diagram showing the lifecycle of *Strongyloides stercoralis*

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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A 23-year-old woman is admitted to the hospital with acute-onset fever, neck stiffness, headache and photophobia. She has no significant past medical history. A CT scan of the head is normal. The patient subsequently undergoes a lumbar puncture which confirms a diagnosis of bacterial meningitis.

She lives with a group of flatmates, including her partner, with whom she has had close contact up until 2 days ago. The patient visited her parents' house and had close contact with them 9 days ago; she visited her grandparents' house 16 days ago and had close contact with them.

What is the most appropriate next step?

- ○ No prophylaxis required for contacts ×
- ○ Prophylaxis required for all flatmates ×
- ○ Prophylaxis required for all flatmates and parents ×
- ○ Prophylaxis required for flatmates, parents and grandparents ×
- ○ Prophylaxis required for partner only ×

Submit answer

Reference ranges

Score: **32%**

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A 23-year-old woman is admitted to the hospital with acute-onset fever, neck stiffness, headache and photophobia. She has no significant past medical history. A CT scan of the head is normal. The patient subsequently undergoes a lumbar puncture which confirms a diagnosis of bacterial meningitis.

She lives with a group of flatmates, including her partner, with whom she has had close contact up until 2 days ago. The patient visited her parents' house and had close contact with them 9 days ago; she visited her grandparents' house 16 days ago and had close contact with them.

What is the most appropriate next step?

No prophylaxis required for contacts	10%
Prophylaxis required for all flatmates	36%
Prophylaxis required for all flatmates and parents	30%
Prophylaxis required for flatmates, parents and grandparents	16%
Prophylaxis required for partner only	8%

People who have been exposed to a patient with confirmed bacterial meningitis should be given prophylactic antibiotics if they have close contact within the seven days before onset

Important for me Less important

Guidance around meningitis prophylaxis is quite clear. Prophylaxis should be offered to household members and those in close contact within the past 7 days. As such, it should be **given to all flatmates** as they have been in close contact up until 2 days prior. Oral ciprofloxacin or rifampicin may be used. Ciprofloxacin is now the drug of choice.

Advising **no prophylaxis** is therefore incorrect.

Similarly, advising **both flatmates and parents** to have prophylaxis is incorrect. Whilst the flatmates should be offered prophylaxis, the parents last had contact over 7 days ago.

Prophylaxis for **flatmates, parents and grandparents** is unwarranted. The parents and grandparents last had contact with the patient more than 7 days ago and so it is not needed for them.

Only advising the partner to have prophylactic antibiotics is incorrect. The entire household has had close contact with her and so all flatmates, not just her partner, should be offered them.

Discuss (7) | Improve

Next question >

Meningitis: management of suspected bacterial meningitis

The investigation and management of suspected bacterial meningitis are usually intertwined given the potential negative impact of delayed antibiotic treatment.

All patients should be transferred to hospital urgently. If patients are in a pre-hospital setting (for example a GP surgery) and meningococcal disease is suspected then intramuscular benzylpenicillin may be given, as long as this doesn't delay transit to hospital.

An ABC approach should be taken initially

- Airway
- Breathing
- Circulation
- Disability: GCS, ; focal neurological signs; seizures; papilloedema;

Senior review if any **warning signs** are present (please see the Meningitis Research Foundation algorithm link for the full list). Examples include:

- rapidly progressive rash
- poor peripheral perfusion
- respiratory rate < 8 or > 30 / min *or* pulse rate < 40 or > 140 / min
- pH < 7.3 *or* WBC< 4 *10^9/L *or* lactate > 4 mmol/L
- GCS < 12 or a drop of 2 points
- poor response to fluid resuscitation

A key decision is when/whether to attempt a lumbar puncture (LP)

- LPs require an aseptic technique and are potentially technical difficult - this means they make take time and delay other treatment
- on the other hand, having a cerebrospinal fluid (CSF) sample can be very useful in confirming the diagnosis and guiding antibiotic treatment
- as always, patient safety means that if there is any doubt IV antibiotics should be given as a priority

Lumbar puncture should be delayed in the following circumstances

- signs of severe sepsis or a rapidly evolving rash
- severe respiratory/cardiac compromise

- significant bleeding risk
- signs of raised intracranial pressure
    - focal neurological signs
    - papilloedema
    - continuous or uncontrolled seizures
    - GCS ≤ 12

Management of patients without indication for delayed LP

- IV access → take bloods and blood cultures
- lumbar puncture
    - if this cannot be done within the first hour, IV antibiotics should be given after blood cultures have been taken
- IV antibiotics
    - 3 months - 50 years: BNF recommends cefotaxime (or ceftriaxone)
    - > 50 years: BNF recommends cefotaxime (or ceftriaxone) + amoxicillin (or ampicillin) for adults
- IV dexamethasone
    - the BNF recommend to '*consider adjunctive treatment with dexamethasone (particularly if pneumococcal meningitis suspected in adults), preferably starting before or with first dose of antibacterial, but no later than 12 hours after starting antibacterial; avoid dexamethasone in septic shock, meningococcal septicaemia, or if immunocompromised, or in meningitis following surgery*'
- CT scan is not normally indicated

Management of patients with signs of raised intracranial pressure

- get critical care input
- secure airway + high-flow oxygen
- IV access → take bloods and blood cultures
- IV dexamethasone
- IV antibiotics as above
- arrange neuroimaging

Management of patients with signs of severe sepsis or a rapidly evolving rash

- get critical care input
- secure airway + high-flow oxygen
- IV access → take bloods and blood cultures
- IV fluid resuscitation
- IV antibiotics as above

Investigations

The bloods that have already been taken should be tested for the following:

- full blood count

- renal function
- glucose
- lactate
- clotting profile
- CRP

If an LP has been performed, the CSF should be tested for:

- glucose, protein, microscopy and culture
- lactate
- meningococcal and pneumococcal PCR
- enteroviral, herpes simplex and varicella-zoster PCR
- consider investigations for TB meningitis

Additional tests that may be helpful include:

- blood gases
- throat swab for meningococcal culture

BNF antibiotic guidelines

Scenario	BNF recommendation
Initial empirical therapy aged < 3 months	IV cefotaxime + amoxicillin (or ampicillin)
Initial empirical therapy aged 3 months - 50 years	IV cefotaxime (or ceftriaxone)
Initial empirical therapy aged > 50 years	IV cefotaxime (or ceftriaxone) + amoxicillin (or ampicillin)
Meningococcal meningitis	IV benzylpenicillin or cefotaxime (or ceftriaxone)
Pneumococcal meningitis	IV cefotaxime (or ceftriaxone)
Meningitis caused by *Haemophilus influenzae*	IV cefotaxime (or ceftriaxone)
Meningitis caused by Listeria	IV amoxicillin (or ampicillin) + gentamicin

If the patient has a history of immediate hypersensitivity reaction to penicillin or to cephalosporins the BNF recommends using chloramphenicol.

Management of contacts

Prophylaxis needs to be offered to households and close contacts of patients affected with meningococcal meningitis. Prophylaxis should also be offered to people who have been exposed

to respiratory secretion, regardless of the closeness of contact. The risk to contacts is highest in the first 7 days but persists for at least 4 weeks.

Management

- people who have been exposed to a patient with confirmed bacterial meningitis should be given prophylactic antibiotics if they have close contact within the 7 days before onset
- oral ciprofloxacin or rifampicin may be used. The Health Protection Agency (HPA) guidelines now state that whilst either may be used ciprofloxacin is the drug of choice as it is widely available and only requires one dose
- meningococcal vaccination should be offered to close contacts when serotype results are available, including booster doses to those who had the vaccine in infancy
- for pneumococcal meningitis, no prophylaxis is generally needed. There are however exceptions to this. If a cluster of cases of pneumococcal meningitis occurs the HPA have a protocol for offering close contacts antibiotic prophylaxis. Please see the link for more details

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

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Links

Meningitis Research Foundation 22 13

Meningitis protocol

BNF 15 9

Medical emergencies in the community

Health Protection Agency 14 7

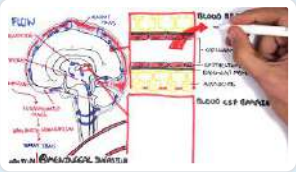
2018 Guidance for public health management of meningococcal disease in the UK

NICE 13 5

2010 Bacterial meningitis and meningococcal septicaemia guidelines

Suggest link Report broken link

Media



(Bacterial) Meningitis Pathophysiology

Armando Hasudungan - YouTube 5 0



Meningitis

Osmosis - YouTube 4 0

Report broken media

Score: **15.8%**

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Back to top

A 35-year-old ex intravenous drug user has been diagnosed with has HBe-Ag positive Hepatitis B. Her investigation results are shown below:

HBV DNA	2100 IU/ml
ALT	60 IU/L

ALT last checked 3 months ago and found to be ALT 60 IU/L.

What is the recommended first line treatment for this patient?

- Tenofovir disoproxil
- Entecavir
- Peginterferon alfa-2a
- Telbivudine
- Sofosbuvir

Submit answer

Reference ranges

Score: **32%**

1 ✓
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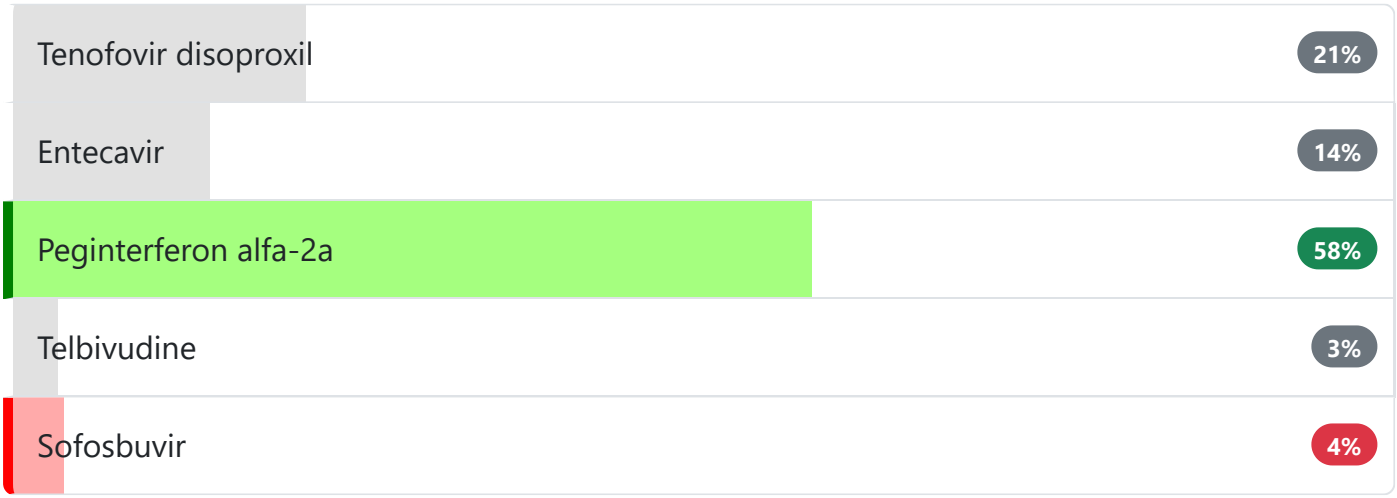
Back to top

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HBV DNA	2100 IU/ml
ALT	60 IU/L

ALT last checked 3 months ago and found to be ALT 60 IU/L.

What is the recommended first line treatment for this patient?



The correct answer here is Peginterferon alfa-2a.

In patients who do not undergo HBeAg seroconversion or who relapse, tenofovir disoproxil is second line. Entecavir is an alternative second line when patients are unable to tolerate tenofovir disoproxil. Telbivudine is no longer recommended for the treatment of chronic Hepatitis B. Sofosbuvir is used for treating hepatitis C.

Please go to the link below for more information:
https:www.nice.org.uk/guidance/CG165/chapter/1-Recommendations

Discuss (9) | Improve

Next question >

Hepatitis B

Hepatitis B is a double-stranded DNA hepadnavirus and is spread through exposure to infected blood or body fluids, including vertical transmission from mother to child. The incubation period is 6-20 weeks.

The features of hepatitis B include fever, jaundice and elevated liver transaminases.

Complications of hepatitis B infection

- chronic hepatitis (5-10%). 'Ground-glass' hepatocytes may be seen on light microscopy
- fulminant liver failure (1%)
- hepatocellular carcinoma
- glomerulonephritis
- polyarteritis nodosa
- cryoglobulinaemia

Immunisation against hepatitis B (please see the Greenbook link for more details)

- children born in the UK are now vaccinated as part of the routine immunisation schedule. This is given at 2, 3 and 4 months of age
- at risk groups who should be vaccinated include: healthcare workers, intravenous drug users, sex workers, close family contacts of an individual with hepatitis B, individuals receiving blood transfusions regularly, chronic kidney disease patients who may soon require renal replacement therapy, prisoners, chronic liver disease patients
- contains HBsAg adsorbed onto aluminium hydroxide adjuvant and is prepared from yeast cells using recombinant DNA technology
- around 10-15% of adults fail to respond or respond poorly to 3 doses of the vaccine. Risk factors include age over 40 years, obesity, smoking, alcohol excess and immunosuppression
- testing for anti-HBs is only recommended for those at risk of occupational exposure (i.e. Healthcare workers) and patients with chronic kidney disease. In these patients anti-HBs levels should be checked 1-4 months after primary immunisation
- the table below shows how to interpret anti-HBs levels:

Anti-HBs level (mIU/ml)	**Response**
> 100	Indicates adequate response, no further testing required. Should still receive booster at 5 years
10 - 100	Suboptimal response - one additional vaccine dose should be given. If immunocompetent no further testing is required
< 10	Non-responder. Test for current or past infection. Give further vaccine course (i.e. 3 doses again) with testing following. If still fails to respond then HBIG would be required for protection if exposed to the virus

Management of hepatitis B

- pegylated interferon-alpha used to be the only treatment available. It reduces viral replication in up to 30% of chronic carriers. A better response is predicted by being female, < 50 years old, low HBV DNA levels, non-Asian, HIV negative, high degree of inflammation on liver biopsy
- whilst NICE still advocate the use of pegylated interferon firstl-line other antiviral medications are increasingly used with an aim to suppress viral replication (not in a dissimilar way to treating HIV patients)
- examples include tenofovir, entecavir and telbivudine (a synthetic thymidine nucleoside analogue)



Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

7 7

2013 Chronic hepatitis B guidelines

Green Book

8 5

Hepatitis B guidelines

Suggest link

Report broken link

Media



Viral hepatitis

Osmosis - YouTube 5 0



Anti-hepatitis medications

Osmosis - YouTube 1 0



Hepatitis

Podmedics - YouTube 1 1

Report broken media

Score: **15.8%**

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A 32-year-old lady presented after returning from Nigeria 3 weeks ago to her GP complaining of feeling feverish and pain in her joints making walking difficult. She denied any rash, or headaches however she has been very drowsy and lethargic in the past week. She spent a week in Nigeria and visited her relatives both in urban and rural areas. She has been taking her malaria prophylaxis tablets. She denies being sexually active for the past year. Her GP sent off three malaria screens which were negative.

Two months later she represents to the emergency department following a seizure and is found to be confused and disorientated. She is irritable, and you notice her hand is trembling. Her sister says she has lost her appetite and weight and has been behaving strangely in the past month. She spends most of her time in bed during the day complaining of a headache and has become withdrawn. On examination, there is hypertonia in her limbs, with hyperreflexia, but the rest of examination is difficult as she is uncooperative.
Investigations:

Na+	140mmol/l
K+	4.3 mmol/l
Urea	5.3 mmol/l
Creatinine	85 µmol/l
Serum bilirubin	15 µmol/l
Serum alkaline phosphatase	85 IU/l
Serum aspartate aminotransferase	19 IU/l
Serum Albumin	25 g/l
C Reactive protein (CRP)	25 mg/l
Erythrocyte Sedimentation Rate (ESR)	75 mm/hr
Haemoglobin	96 g/l
White cell count	10.2 x 10^9/L
Neutrophils	8.9 x 10^9/L
Eosinophilia	0.1 x10^9/L
Lymphocytes	1.8 x 10^9/L
Platelets	140 x 10^9/L
INR	1.0

What is the likely causative organism?

- *Plasmodium vivax*
- *Borrelia burgdorferi*
- *Trypanosoma gambiense*
- *Trypanosoma rhodesiense*
- *Trypanosoma cruzi*

Submit answer

Reference ranges

Score: **32%**

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C Reactive protein (CRP)	25 mg/l
Erythrocyte Sedimentation Rate (ESR)	75 mm/hr
Haemoglobin	96 g/l
White cell count	10.2 x 10^9/L
Neutrophils	8.9 x 10^9/L
Eosinophilia	0.1 x10^9/L
Lymphocytes	1.8 x 10^9/L
Platelets	140 x 10^9/L
INR	1.0

What is the likely causative organism?

Plasmodium vivax	5%
Borrelia burgdorferi	8%
Trypanosoma gambiense	55%
Trypanosoma rhodesiense	21%
Trypanosoma cruzi	12%

This lady of African background presents initially with fever after travelling from Nigeria and goes on to develop chronic symptoms of daytime somnolence, continuous headaches and behavioural changes. These symptoms suggest African Sleeping Sickness or African Trypanosomiasis. *Trypanosoma gambiense* is more common than *Trypanosoma rhodesiense* in patients from West Africa, such as Nigeria.

Early stages of the disease tends to present about 3 weeks after being bitten by an infected tsetse fly. Symptoms include fevers, arthralgia, lethargy, drowsiness. Late stages include behavioural and mood changes, headache and the pathognomonic daytime somnolence with nocturnal insomnia.

Borrelia burgdorferi is transmitted by ticks (Ixodes spp.) and causes Lyme disease. They occur in the USA, northern and central European countries, and temperate forested areas of Asia.

Plasmodium vivax is one species of Plasmodium, the parasite that causes malaria. It is spread by infected mosquitoes. It is an important differential of fever in the returning traveller, however as the initial malaria screens were negative, and the central nervous system symptoms in the late stages are not consistent with a malarial infection.

Trypanosoma cruzi is responsible for American Trypanosomiasis (or Chagas' disease). It is transmitted by contamination with infected faeces of assassin bugs (reduviid bugs). It is found in South America and the south USA. Chronic infection can lead to chronic congestive heart failure, megaoesophagus and megacolon.

Discuss (11) Improve

Next question ›

Trypanosomiasis ★

Two main form of this protozoal disease are recognised - African trypanosomiasis (sleeping sickness) and American trypanosomiasis (Chagas' disease).

Two forms of **African trypanosomiasis**, or **sleeping sickness**, are seen - *Trypanosoma gambiense* in West Africa and *Trypanosoma rhodesiense* in East Africa. Both types are spread by the tsetse fly. *Trypanosoma rhodesiense* tends to follow a more acute course. Clinical features include:

- Trypanosoma chancre - painless subcutaneous nodule at site of infection
- intermittent fever
- enlargement of posterior cervical lymph nodes
- later: central nervous system involvement e.g. somnolence, headaches, mood changes, meningoencephalitis

Management

- early disease: IV pentamidine or suramin
- later disease or central nervous system involvement: IV melarsoprol

American trypanosomiasis, or **Chagas' disease**, is caused by the protozoan *Trypanosoma cruzi*. The vast majority of patients (95%) are asymptomatic in the acute phase although a chagoma (an erythematous nodule at site of infection) and periorbital oedema are sometimes seen. Chronic Chagas' disease mainly affects the heart and gastrointestinal tract

- myocarditis may lead to dilated cardiomyopathy (with apical atophy) and arrhythmias
- gastrointestinal features includes megaoesophagus and megacolon causing dysphagia and constipation

Management

- treatment is most effective in the acute phase using azole or nitroderivatives such as benznidazole or nifurtimox
- chronic disease management involves treating the complications e.g., heart failure

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

8 3

Chaga's disease

Suggest link

Report broken link

Media



Sleeping Sickness - an introduction to African Trypanosomiasis

Let's Learn About Bugs - YouTube

7 0



Chagas Disease - An overview

Let's Learn About Bugs - YouTube

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Chagas disease

Osmosis - YouTube

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Score: **15.8%**

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A 42-year-old immigrant from Nigeria has been referred to the infectious disease clinic. He was feeling tired and generally unwell over several weeks since last visiting Nigeria, his country of origin. He has also travelled for business to the United States six months ago. He has had fever and rigors as well as headaches. Posterior lymph nodes have been present and have been aspirated one week ago, and trypomastigotes were present. What is the most likely causative organism?

- Trypanosoma rhodesiense
- Trypanosoma gambiense
- Trypanosoma cruzi
- Leishmania mexicana
- Leishmania tropica

Submit answer

Reference ranges

Score: **32%**

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Trypanosoma rhodesiense	20%
Trypanosoma gambiense	56%
Trypanosoma cruzi	20%
Leishmania mexicana	1%
Leishmania tropica	3%

The correct answer is Trypanosoma gambiense. This is a patient with a clinical history suggestive of African trypanosomiasis with a slow wasting course. The presence of trypomastigotes means that there will be a Trypanosoma infection. The species can be determined by geography. Rhodesiense occurs in east Africa whilst gambiense occurs in west Africa, including Nigeria. Uganda is the only country with both. Cruzi is endemic in South America and causes American trypanosomiasis. Leishmania are other protozoa occurring in Africa, South America and India.

Discuss (16) | Improve

Next question

Trypanosomiasis

Two main form of this protozoal disease are recognised - African trypanosomiasis (sleeping sickness) and American trypanosomiasis (Chagas' disease).

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- early disease: IV pentamidine or suramin
- later disease or central nervous system involvement: IV melarsoprol

American trypanosomiasis, or **Chagas' disease**, is caused by the protozoan *Trypanosoma cruzi*. The vast majority of patients (95%) are asymptomatic in the acute phase although a chagoma (an erythematous nodule at site of infection) and periorbital oedema are sometimes seen. Chronic Chagas' disease mainly affects the heart and gastrointestinal tract
- myocarditis may lead to dilated cardiomyopathy (with apical atophy) and arrhythmias
- gastrointestinal features includes megaoesophagus and megacolon causing dysphagia and constipation

Management
- treatment is most effective in the acute phase using azole or nitroderivatives such as benznidazole or nifurtimox
- chronic disease management involves treating the complications e.g., heart failure

AI + Q 123

Next question >

B I A T↕ 123

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

8 3

Chaga's disease

Suggest link Report broken link

Media



Sleeping Sickness - an introduction to African Trypanosomiasis

Let's Learn About Bugs - YouTube 7 0



Chagas Disease - An overview

Let's Learn About Bugs - YouTube 3 1



Chagas disease

Osmosis - YouTube 2 1

Report broken media

Score: **15.8%**

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Back to top

A 23-year-old gentleman presents to GUM clinic. He has been referred by his GP as he presented with dysuria and a negative urine dipstick test. A sexual health screen, including blood tests for HIV and syphilis serology, are requested. What form of consent is required?

- No consent
- Implied consent
- Verbal consent
- Written consent
- Written consent for HIV testing and verbal consent for sexual health screening

Submit answer

Reference ranges

Score: **32%**

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Back to top

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No consent	8%
Implied consent	7%
Verbal consent	50%
Written consent	10%
Written consent for HIV testing and verbal consent for sexual health screening	26%

The correct answer is verbal consent. Consent clearly is needed as an investigation is done but since syphilis and HIV are transmittable diseases consent needs to be specific towards each. This means that the patient must be aware that they are being tested for HIV and syphilis rather than just aware that they are having a routine blood test. Written consent is not needed, and may discourage patients from having testing.

Source:
UK National Guidelines for HIV Testing 2008. London: British HIV Association, 2008
(see page 10)

Discuss (6) | Improve

Next question

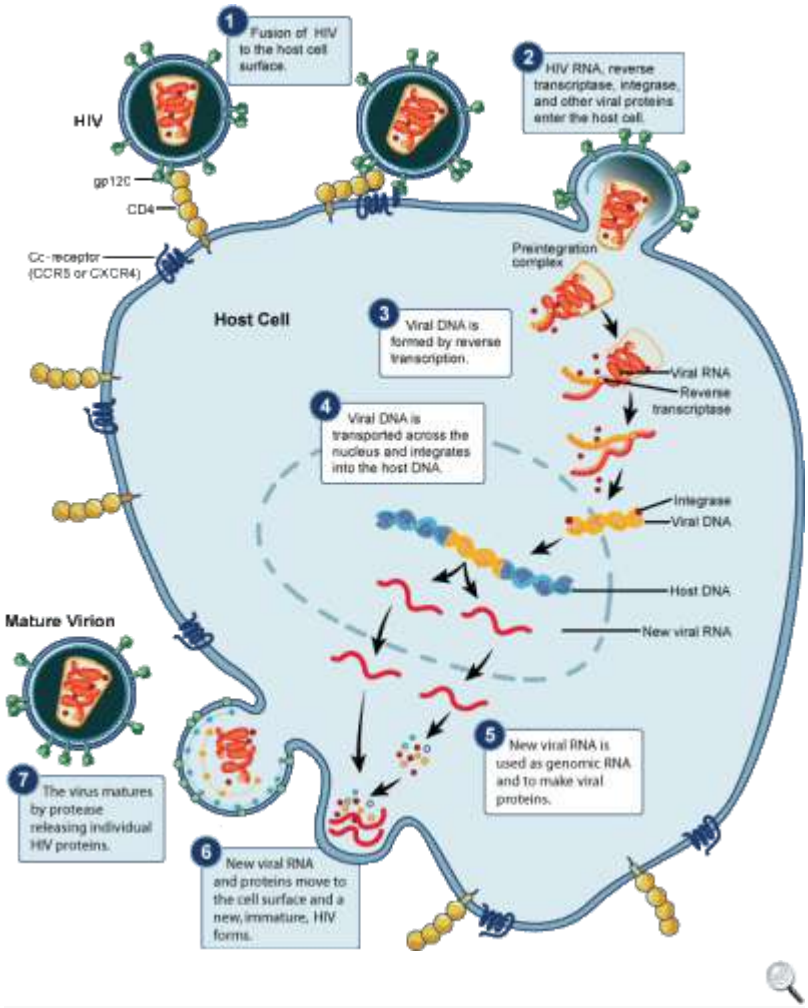
HIV: seroconversion

HIV seroconversion is symptomatic in 60-80% of patients and typically presents as a glandular fever-type illness. Increased symptomatic severity is associated with poorer long-term prognosis. It typically occurs 3-12 weeks after infection

Features

- sore throat

- lymphadenopathy
- malaise, myalgia, arthralgia
- diarrhoea
- maculopapular rash
- mouth ulcers
- rarely meningoencephalitis



An illustration model of the HIV Replication Cycle. Each step of the cycle is numbered and concisely described. Credit: NIAID

123

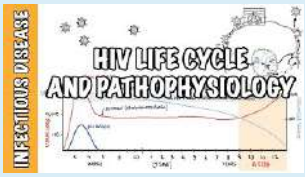
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Human immunodeficiency virus (HIV) - life cycle, pathophysiology, investigations, diagnosis

Armando Hasudungan - YouTube

2 0



HIV

Osmosis - YouTube

4 2

Report broken media

Score: **15.8%**

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Back to top

A 30-year-old man returns from a stag do in Amsterdam with fever, headache and abdominal pain. He was away for 2 days and drunk 40-50 units of alcohol. He also had unprotected sex with a local girl and vaguely remembers falling in the canal on the second night. He is febrile with a blood pressure 100/70 mmHg. His bloods tests reveal :

Routine tests in the emergency department are ordered:

Hb 95 g/l WBC 20 10^9/l , Platelets 150 10^9/l

Na^+ 135 mmol/l , K^+ 5.0 mmol/l

Urea 15 mmol/l , Creatinine 300 µmol/l

Bilirubin 35 µmol/l , ALP 200 u/l , ALT 70 u/l

CXR - pulmonary haemorrhage

What is the most likely diagnosis?

- Acute hepatitis A
- Acute hepatitis B
- Goodpastures syndrome
- Leptospirosis
- Atypical pneumonia

Submit answer

Reference ranges

Score: **32%**

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Back to top

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Hb 95 g/l WBC 20 10^9/l , Platelets 150 10^9/l

Na^+ 135 mmol/l , K^+ 5.0 mmol/l

Urea 15 mmol/l , Creatinine 300 µmol/l

Bilirubin 35 µmol/l , ALP 200 u/l , ALT 70 u/l

CXR - pulmonary haemorrhage

What is the most likely diagnosis?

Acute hepatitis A	2%
Acute hepatitis B	1%
Goodpastures syndrome	10%
Leptospirosis	83%
Atypical pneumonia	4%

This gentleman should be screened for viral hepatitis and have an HIV test, however given the results of his investigations infection with Leptospira from falling in the contaminated water is the most likely diagnosis. The acuity and neutrophilia go against Goodpasture's syndrome and an atypical pneumonia would not classically cause pulmonary haemorrhage.

Leptospirosis

Leptospirosis is caused by the spirochaete *Leptospira interrogans* (serogroup *L. icterohaemorrhagiae*), classically being spread by contact with infected rat urine.

Epidemiology

- leptospirosis is commonly seen in questions referring to sewage workers, farmers, vets or people who work in an abattoir
- however, on an international level, leptospirosis is far more common in the tropics so should be considered in the returning traveller

Weil's disease should always be considered in high-risk patients with hepatorenal failure

Features

- the early phase is due to bacteraemia and lasts around a week
  - may be mild or subclinical
  - fever
  - flu-like symptoms
  - subconjunctival suffusion (redness)/haemorrhage
- second immune phase may lead to more severe disease (Weil's disease)
  - acute kidney injury (seen in 50% of patients)
  - hepatitis: jaundice, hepatomegaly
  - aseptic meningitis

Investigation

- **serology**: antibodies to *Leptospira* develop after about 7 days
- PCR
- culture
  - growth may take several weeks so limits usefulness in diagnosis
  - blood and CSF samples are generally positive for the first 10 days
  - urine cultures become positive during the second week of illness

Management

- high-dose benzylpenicillin or doxycycline

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Quarterly Journal of Medicine 12 4

Leptospirosis review

Suggest link Report broken link

Media



Leptospira

Osmosis - YouTube 7 0

Report broken media

Score: **15.8%**

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A 68-year-old female was admitted to the ward with left arm cellulitis. She was septic on admission and the sepsis 6 bundle was completed in one hour. She is treated with IV flucloxacillin initially and was switched to oral flucloxacillin on day 2. On day 3 of admission you receive a call from the duty microbiologist with the following results:

Anaerobic blood culture bottle	Positive for *Staphylococcus aureus*
Aerobic blood culture bottle	Positive for *Staphylococcus aureus*

How will you manage this patient?

- ○ IV vancomycin ×
- ○ Oral linezolid ×
- ○ IV flucloxacillin for two weeks ×
- ○ Oral flucloxacillin for two weeks ×
- ○ Oral flucloxacillin for one week ×

Submit answer

Reference ranges

Score: **32%**

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Anaerobic blood culture bottle	Positive for *Staphylococcus aureus*
Aerobic blood culture bottle	Positive for *Staphylococcus aureus*

How will you manage this patient?

- IV vancomycin — 12%
- Oral linezolid — 4%
- IV flucloxacillin for two weeks — 32%
- Oral flucloxacillin for two weeks — 37%
- Oral flucloxacillin for one week — 15%

Staphylococcus aureus bacteraemia (SAB) is a serious condition which may occur secondary to soft tissue, joint, bone, indwelling IV line or cardiac infection. It is treated with a minimum of two weeks IV flucloxacillin

Important for me Less important

Staphylococcus aureus bacteraemia (SAB) is a serious condition with significant morbidity and mortality. SAB is generally caused by soft tissue, joint, bone, indwelling IV line or cardiac infections. Once the bacteria has entered the bloodstream, they are particularly effective at infecting various organs, potentially causing infective endocarditis, septic arthritis, discitis, and osteomyelitis.

Treatment is with intravenous high dose (2 grams) flucloxacillin for a minimum of two weeks. An MRSA risk assessment should be performed. If the patient is at high risk of MRSA then they should be treated with vancomycin instead.

It is important to look for sources of infection and to consider investigating for sequelae (e.g. transthoracic echo to assess for endocarditis).

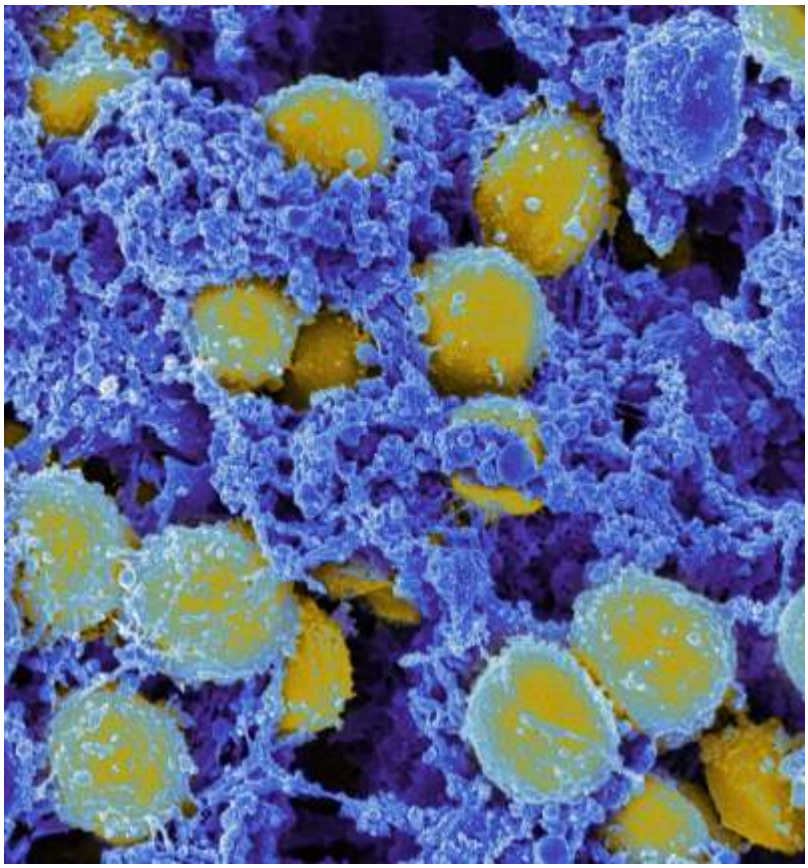
Staphylococci

Staphylococci are a common type of bacteria that are often found normal commensal organisms but may also cause invasive disease. Some basic facts include:

- Gram-positive cocci
- facultative anaerobes
- produce catalase

The two main types of Staphylococci you need to know about are *Staphylococcus aureus* and *Staphylococcus epidermidis*.

Staphylococcus aureus	***Staphylococcus epidermidis***
• Coagulase-positive • Causes skin infections (e.g. cellulitis), abscesses, osteomyelitis, toxic shock syndrome	• Coagulase-negative • Cause of central line infections and infective endocarditis



Scanning electromicrograph of Staphylococcus aureus bacteria. Credit: NIAID

Next question

Textbooks

High-yield textbook

Extended textbook

Media



Staphylococcus species (Characteristics, Clinical complications, Differences)

ATP - YouTube

7 0

Report broken media

Score: **15.8%**

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A 34-year-old female attended the emergency department with a rash. Her past medical history was unremarkable apart from being on the oral contraceptive pill. She had been recovering from a widespread, pruritic, vesicular rash over the past 8 days after coming into contact with her nephew with chickenpox. Yesterday she began to feel lethargic and unwell, with a high temperature and a painful left calf. This morning she had noticed an area of erythema on her lower leg which seemed to get larger and more painful throughout the day.

On examination, she looked unwell. There were healed varicella scars and a few crusted scabs present over her lower limbs and torso. There was a diffuse erythematous rash extending from her mid-calf to her ankle. The area was oedematous and very tender. She had a normal range of movement in all joints of the leg, albeit limited by pain.

Her observations were as follows:

- temperature 39.7ºC
- respiratory rate 24/min
- blood pressure 91/55mmHg
- heart rate 112bpm
- oxygen saturations 98% on room air

Her blood results were as follows:

Hb	109 g/L	115 - 160
WBC	20 * 10^9/L	(4.0 - 11.0)
CRP	208 mg/L	(< 5)

She was started on intravenous fluids and given analgesia. Her IV morphine was titrated up to control her pain and she was started on broad-spectrum antibiotics.

The following morning she remained tachypnoeic and tachycardic. The area of erythema now extended proximally to 2cm below her knee. A small area of dark purple-black discolouration was present at the distal aspect of the rash.

What is the next most appropriate step in management?

- IV aciclovir
- Plasmapheresis
- MRI lower leg

- Surgical debridement
- Treatment dose low molecular weight heparin

Submit answer

Reference ranges

Score: **32%**

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What is the next most appropriate step in management?

IV aciclovir	10%
Plasmapheresis	3%
MRI lower leg	4%

Surgical debridement 78%

Treatment dose low molecular weight heparin 5%

Chickenpox is a risk factor for invasive group A streptococcal soft tissue infections including necrotizing fasciitis

Important for me Less important

This is a case of bacterial superinfection complicating acute varicella infection. The systemic involvement and rapid spread of cellulitis despite broad-spectrum antibiotics should alert you to the possible diagnosis of necrotising fasciitis. Whilst the most common type of necrotising fasciitis is type 1 (caused by polymicrobial infection), most cases of post-varicella necrotising fasciitis are caused by a monomicrobial infection by group A streptococcus (e.g. *Streptococcus pyogenes*).

Bacteria are introduced into the skin via skin trauma (in this case, the varicella skin lesions formed a portal of entry) and track along fascial planes, extending beyond the area of overlying cellulitis.

The definite management of necrotising fasciitis is surgical debridement of all infected tissue. As the infection progresses, necrosis and hypoperfusion of the area limit the delivery of antibiotics to the area. Without debridement, mortality is 100% and any delay increases the risk of death.

Whilst IV aciclovir is important in the treatment of acute varicella infection, the most important next step is to remove the source of the bacterial infection. Chickenpox has contributed to this presentation but is not the cause of her acute deterioration or necrotic skin.

Whilst MRI can confirm the extent of necrotising fasciitis, it may delay surgical debridement. This patient is clearly unwell and needs immediate definitive management.

Treatment dose low molecular weight heparin would be appropriate if the cause was a deep vein thrombosis but this presentation is more in keeping with a rapidly progressive bacterial infection.

Plasmapheresis does not have a role in the management of necrotising fasciitis.

Discuss Improve

Next question

Chickenpox

Chickenpox is caused by primary infection with varicella zoster virus. Shingles is a reactivation of the dormant virus in dorsal root ganglion

Chickenpox is highly infectious

- spread via the respiratory route
- can be caught from someone with shingles
- infectivity = 4 days before rash, until 5 days after the rash first appeared*
- incubation period = 10-21 days

Clinical features (tend to be more severe in older children/adults)

- fever initially
- itchy, rash starting on head/trunk before spreading. Initially macular then papular then vesicular
- systemic upset is usually mild

Management is supportive

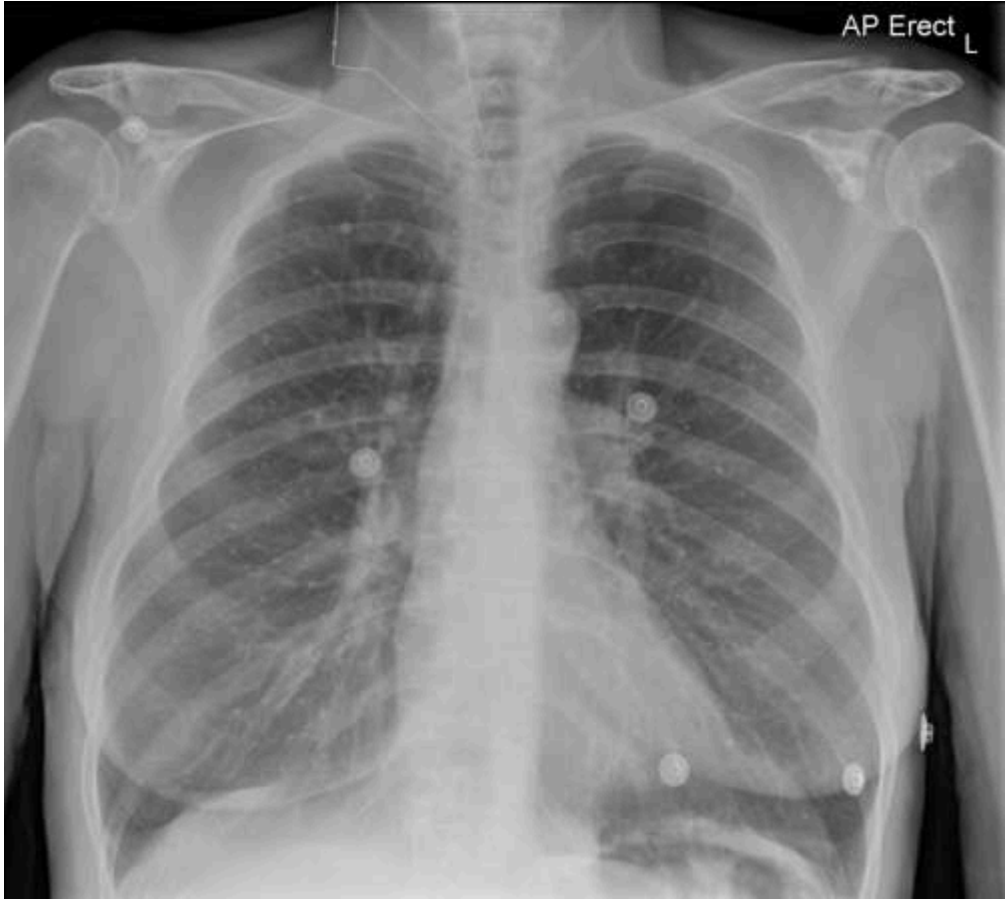
- keep cool, trim nails
- calamine lotion
- school exclusion: NICE state the following: *Advise that the most infectious period is 1-2 days before the rash appears, but infectivity continues until all the lesions are dry and have crusted over (usually about 5 days after the onset of the rash).*
- immunocompromised patients and newborns with peripartum exposure should receive varicella zoster immunoglobulin (VZIG). If chickenpox develops then IV aciclovir should be considered

A common complication is secondary bacterial infection of the lesions

- NSAIDs may increase this risk
- whilst this commonly may manifest as a single infected lesion/small area of cellulitis, in a small number of patients invasive group A streptococcal soft tissue infections may occur resulting in necrotizing fasciitis

Rare complications include

- pneumonia
- encephalitis (cerebellar involvement may be seen)
- disseminated haemorrhagic chickenpox
- arthritis, nephritis and pancreatitis may very rarely be seen



© Image used on license from Radiopaedia

Chest x-ray showing miliary opacities secondary to healed varicella pneumonia. Multiple tiny calcific miliary opacities noted throughout both lungs. These are of uniform size and dense suggesting calcification. There is no focal lung parenchymal mass or cavitating lesion seen. The appearances are characteristic for healed varicella pneumonia.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Greenbook 8 6

Varicella

Public Health England 8 6

Chapter 9: managing specific infectious diseases

Suggest link Report broken link

Media



Varicella zoster virus

Osmosis - YouTube 6 0

Report broken media

Score: **15.8%**

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Back to top

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On examination, he has a fever of 39°C and a pulse of 135bpm. His blood pressure is 100/70mmHg. He is icteric but does not have any flapping tremors. There is evidence of an enlarged spleen which is palpable 3 finger breadths below the left costal margin. The liver span is normal.

Lab reports reveal:

Hb	115 g/l
Platelets	100 * 10^9/l
WBC	9.5 * 10^9/l
Reticulocytes	5% (0.2 - 2%)

Na^+	140 mmol/l
K^+	4.6 mmol/l
Urea	5.1 mmol/l
Creatinine	83 µmol/l

Bilirubin	49 µmol/l
AST	50 u/l
ALT	25 u/l
Glucose	6.0 mmol/l

CT scan brain: Normal

CSF examination reveals:

Appearance	Clear
Protein	0.3 g/L (0.2 0.4 g/L)
Glucose	5.3 mmol/l
Lymphocytes	15/mm^3

Neutrophils	10

Which of the following is the most appropriate treatment option?

- Chloroquine
- Artemether/lumefantrine
- Primaquine
- IV acyclovir
- Co-trimoxazole

Submit answer

Reference ranges

Score: **32%**

1 ✓
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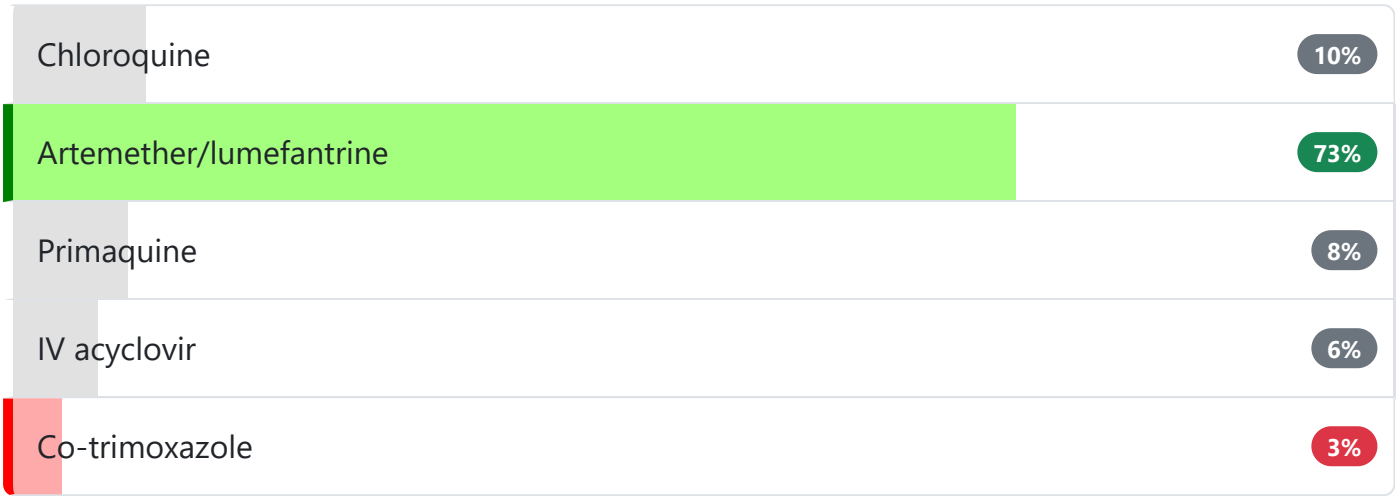
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Lymphocytes	15/mm³

Neutrophils	10

Which of the following is the most appropriate treatment option?



The high-grade fever, history of travel to an endemic area, splenomegaly, thrombocytopaenia, evidence of haemolysis (raised bilirubin and reticulocytosis) and normal blood leucocyte count all favour a diagnosis of cerebral malaria

The treatment modality most suited would be a combination of artemether and lumefantrine which would provide adequate coverage from plasmodium falciparum.

Diagnosis is through examination of blood films (thick and thin films) for evidence of parasitaemia and the ICT-MP.

The CSF findings are non-specific with the only abnormality being a mild lymphocytosis which is of no bearing on the scenario. It, however, rules out bacterial or tuberculous meningitis.

The mildly raised AST is in keeping with his history of alcohol intake.

Discuss (12) | Improve

Next question >

Malaria: Falciparum ★

Falciparum malaria is the commonest, and most severe, type of malaria.

Clinical features

Falciparum malaria typically presents with symptoms suggestive of a flu-like illness. The classical triad of symptoms includes paroxysms of fever, chills, and sweating. These symptoms may occur every 48 hours corresponding to the erythrocytic cycle of the *Plasmodium falciparum* parasite.

Fever is often high, intermittent, and may be accompanied by rigors. Initial manifestations can be non-specific and include malaise, headache, and myalgia, which might be mistaken for a viral syndrome.

General features of falciparum malaria

- fever: The hallmark of malaria, typically cyclical, often accompanied by sweating and sometimes rigors.
- gastrointestinal:
  - anorexia, nausea, vomiting, and abdominal pain are frequent
  - diarrhoea can occasionally be a feature, more commonly in children
  - patients may exhibit mild jaundice and occasional pruritus
- respiratory: Cough and, in some cases, mild tachypnoea may occur, though primary respiratory complications are rare in non-severe cases.
- musculoskeletal: Generalised body aches and joint pains are common.
- neurological:
  - headache is prominent and often severe.
  - dizziness and sleep disturbances may also be observed.
- cardiovascular: Tachycardia may be evident, and although hypotension is more typical of severe malaria, it can occasionally be seen in non-severe cases as well.
- haematological:
  - thrombocytopaenia is the most significant haematological finding and can occur in the absence of severe disease.
  - mild anaemia may also be present.
- renal: While acute kidney injury is associated with severe malaria, non-severe cases might show mild to moderate increases in creatinine or blood urea nitrogen levels.

Feature of severe falciparum malaria

- schizonts on a blood film
- parasitaemia > 2%
- hypoglycaemia
- acidosis
- temperature > 39 °C
- severe anaemia
- complications as below

Complications

- cerebral malaria: seizures, coma
- acute renal failure: blackwater fever, secondary to intravascular haemolysis, mechanism unknown
- acute respiratory distress syndrome (ARDS)
- hypoglycaemia
- disseminated intravascular coagulation (DIC)

Management

Uncomplicated falciparum malaria

- strains resistant to chloroquine are prevalent in certain areas of Asia and Africa
- the 2010 WHO guidelines recommend artemisinin-based combination therapies (ACTs) as first-line therapy
- examples include artemether plus lumefantrine, artesunate plus amodiaquine, artesunate plus mefloquine, artesunate plus sulfadoxine-pyrimethamine, dihydroartemisinin plus piperaquine

Severe falciparum malaria

- a parasite counts of more than 2% will usually need parenteral treatment irrespective of clinical state
- intravenous artesunate is now recommended by WHO in preference to intravenous quinine
- if parasite count > 10% then exchange transfusion should be considered
- shock may indicate coexistent bacterial septicaemia - malaria rarely causes haemodynamic collapse



Next question

Textbooks

High-yield textbook

Extended textbook

Links

WHO

9 7

Malaria treatment guidelines

Suggest link Report broken link

Media



Malaria

Osmosis - YouTube 5 1

Report broken media

Score: **15.8%**

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A 38-year-old woman presents to her HIV clinic for her 6-monthly review having had her routine blood tests two weeks prior to her clinic visit. She has diagnosed with HIV seven years ago and has been stable on antiretroviral therapy since then. Her other medical problems include asthma which is well controlled. She reports generally good compliance with her medications but has unfortunately missed two of her treatment doses as she was on a weekend holiday and forgot to take her tablets with her. She feels well in herself.

Blood tests:

	Today	Two weeks ago	One year ago
HIV viral load	110 copies/ml	<50 copies/ml	<50 copies/ml
CD4 count	983 cells/mm^3	912 cells/mm^3	Not tested

How should she be further investigated?

- Urgent CD4 count
- Urgent viral resistance testing
- Urgent HIV-1 and HIV-2 serology
- Repeat viral load in one month
- Routine viral load and CD4 count in six months

Submit answer

Reference ranges

Score: **32%**

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Blood tests:

	Today	Two weeks ago	One year ago
HIV viral load	110 copies/ml	<50 copies/ml	<50 copies/ml
CD4 count	983 cells/mm^3	912 cells/mm^3	Not tested

How should she be further investigated?

Urgent CD4 count	3%
Urgent viral resistance testing	19%
Urgent HIV-1 and HIV-2 serology	5%
Repeat viral load in one month	47%
Routine viral load and CD4 count in six months	27%

The question describes a possible viral blip, a single viral load that is detectable but less than 200 copies/ml. This is a common feature in chronic HIV management and does not imply failure of treatment. However, good compliance should be reinforced and viral load should be repeated within one month so exclude virological failure or rebound. A CD4 count would not be reassuring in this circumstance as if normal there could still be a rebound happening. Serology only aids diagnosis. Viral resistance testing is useful in order to determine the cause of virological failure. Routine monitoring is not appropriate as a viral rebound will need to be excluded.

HIV monitoring

HIV, once stable, should be monitored with a viral load every six months and CD4 counts annually at least once virological suppression, defined as sustained viral load less than <50 copies/ml, is established. If the viral load increases to between 50-200 copies/ml on a single sample this is defined as a viral blip and is unlikely to be significant, but a viral load should be re-tested within one month. If a viral load above >200 copies/ml is sustained this is a virological rebound and can

imply failure. This can be due to new viral resistance, and, if suspected, would need resistance testing.

Source:

'BHIVA Guidelines for the Treatment of HIV-1-positive Adults with Antiretroviral Therapy 2015.' BHIVA. Aug. 2016.

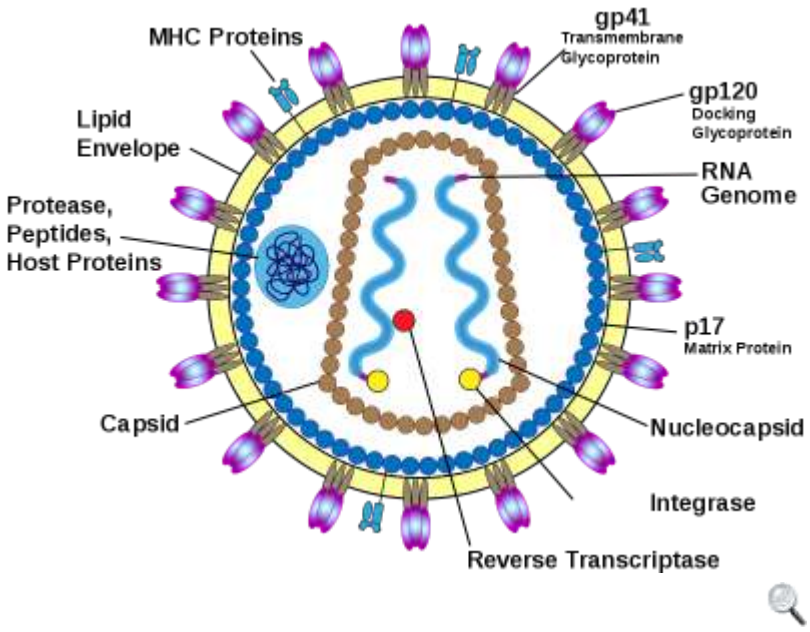
Discuss (5) Improve

Next question ›

HIV: the virus ★

Basics

- HIV is a RNA retrovirus of the lentivirus genus (lentiviruses are characterized by a long incubation period)
- two variants - HIV-1 and HIV-2
- HIV-2 is more common in west Africa, has a lower transmission rate and is thought to be less pathogenic with a slower progression to AIDS



Basics structure

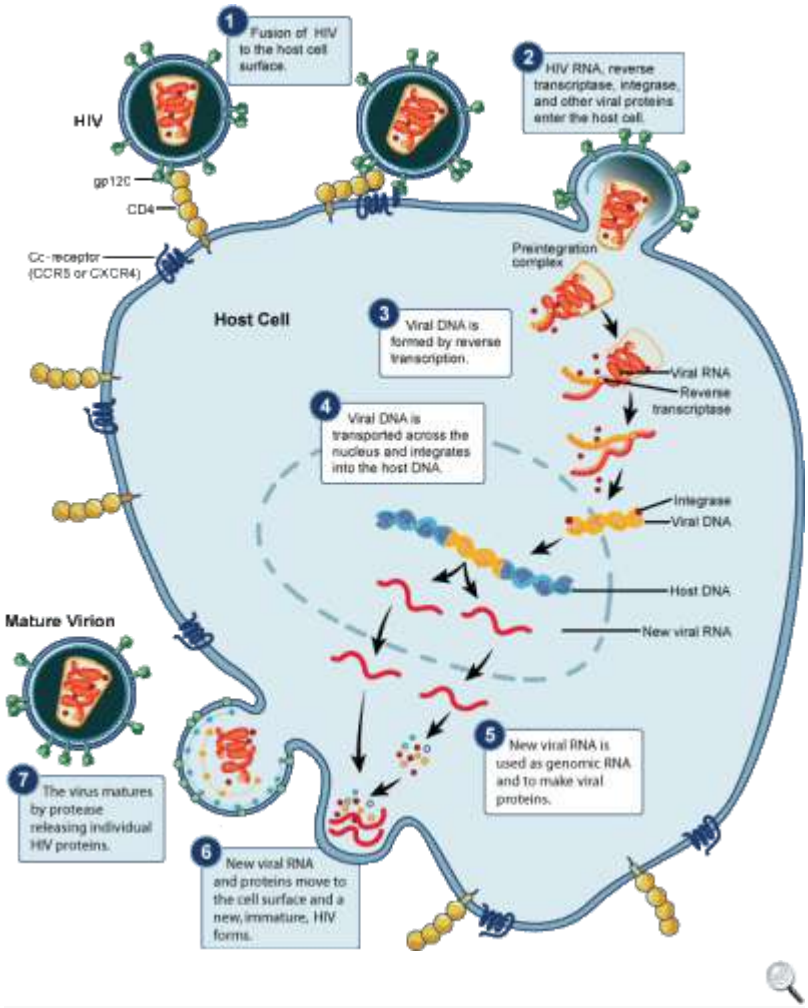
- spherical in shape with two copies of single-stranded RNA enclosed by a capsid of the viral protein p24
- a matrix composed of viral protein p17 surrounds the capsid
- envelope proteins: gp120 and gp41
- pol gene encodes for viral enzymes reverse transcriptase, integrase and HIV protease

Cell entry

- HIV can infect CD4 T cells, macrophages and dendritic cells
- gp120 binds to CD4 and CXCR4 on T cells and CD4 and CCR5 on macrophages
- mutations in CCR5 can give immunity to HIV

Replication

- after entering a cell the enzyme reverse transcriptase creates dsDNA from the RNA for integration into the host cell's genome



An illustration model of the HIV Replication Cycle. Each step of the cycle is numbered and concisely described. Credit: NIAID

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

National Institute for Health

1 0

HIV virus life cycle and sites of actions of anti HIV agents

Suggest link

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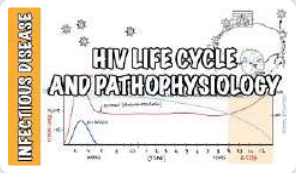
Media



HIV

Osmosis - YouTube

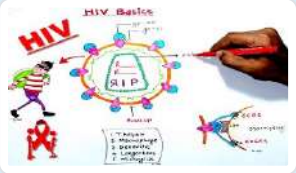
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Human immunodeficiency virus (HIV) - life cycle, pathophysiology, investigations, diagnosis

Armando Hasudungan - YouTube

0 1



HIV basic structure

Medinaz - YouTube

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Score: **15.8%**

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On examination his throat is hyperaemic, but there is no cervical lymphadenopathy. Chest auscultation and percussion reveal no abnormalities. You note dusky red, target shaped skin lesions over all four extremities. Chest x-ray reveals interstitial infiltrates in the left lower lobe. Sputum Gram stain reveals polymorphonuclear cells but no organisms.

Which of the following organisms is most likely responsible for this presentation?

- *Streptococcus pneumoniae* ×
- *Haemophilus influenzae* ×
- *Legionella pneumophila* ×
- *Mycoplasma pneumoniae* ×
- Epstein Barr virus ×

Submit answer

Reference ranges

Score: **32%**

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Which of the following organisms is most likely responsible for this presentation?

Streptococcus pneumoniae	6%
Haemophilus influenzae	2%
Legionella pneumophila	4%
Mycoplasma pneumoniae	80%
Epstein Barr virus	8%

Stereotypical history of mycoplasma pneumonia: worsening flu-like symptoms and a dry cough. Erythema multiforme is noted on examination

Important for me Less important

This young man presents with a history suggestive of atypical pneumonia. Atypical pneumonia is distinguished from typical pneumonia by its more indolent course, non-productive cough and higher incidence of extra-pulmonary symptoms. This patients low grade fever and weeks worth of symptoms are consistent with an indolent course. Furthermore, he has multiple extra-pulmonary symptoms including headache, sore throat and skin rash. The different types of atypical pneumonia can present similarly and are at times challenging to distinguish. In this case, the skin rash (erythema multiforme) is typical of *Mycoplasma*. *Mycoplasma* is an organism that does not have a cell wall, and therefore does not stain with Gram stain; hence the finding of only polymorphonuclear cells on sputum Gram stain analysis.

Choice 1: *Pneumococcus* is the most frequent cause of community-acquired pneumonia requiring hospitalisation. Cough is often productive of rusty-coloured sputum. Compared to atypical pneumonias, pneumococcal pneumonia causes fewer extra-pulmonary manifestations but has a more virulent course. Sputum Gram stain would revel Gram positive diplococci.

Choice 2: *Haemophilus influenzae* is another common cause of community-acquired pneumonia. It presents like a typical pneumonia. Sputum Gram stain reveals small Gram negative bacilli.

Choice 3: *Legionella pneumophila* causes pneumonia with extra-pulmonary symptoms. Environmental water sources are often involved in its spread. It occurs most frequently in elderly patients and smokers. The urine *Legionella antigen* test is helpful in making the diagnosis.

Choice 5: Epstein Barr virus causes infectious mononucleosis, common in adolescents. Sore throat is the most common presenting symptom. Fatigue, malaise, headache and abdominal pain are also common. Concomitant pneumonia is not usually seen.

Discuss (2) Improve

Next question >

Mycoplasma pneumoniae

Mycoplasma pneumoniae is a cause of atypical pneumonia which often affects younger patients. It is associated with a number of characteristic complications such as erythema multiforme and cold autoimmune haemolytic anaemia. Epidemics of *Mycoplasma pneumoniae* classically occur every 4 years. It is important to recognise atypical pneumonia as it may not respond to penicillins or cephalosporins due to it lacking a peptidoglycan cell wall.

Features

- the disease typically has a prolonged and gradual onset
- flu-like symptoms classically precede a dry cough
- bilateral consolidation on x-ray
- complications may occur as below

Complications

- cold agglutins (IgM): may cause an haemolytic anaemia, thrombocytopenia
- erythema multiforme, erythema nodosum
- meningoencephalitis, Guillain-Barre syndrome and other immune-mediated neurological diseases
- bullous myringitis: painful vesicles on the tympanic membrane
- pericarditis/myocarditis
- gastrointestinal: hepatitis, pancreatitis
- renal: acute glomerulonephritis

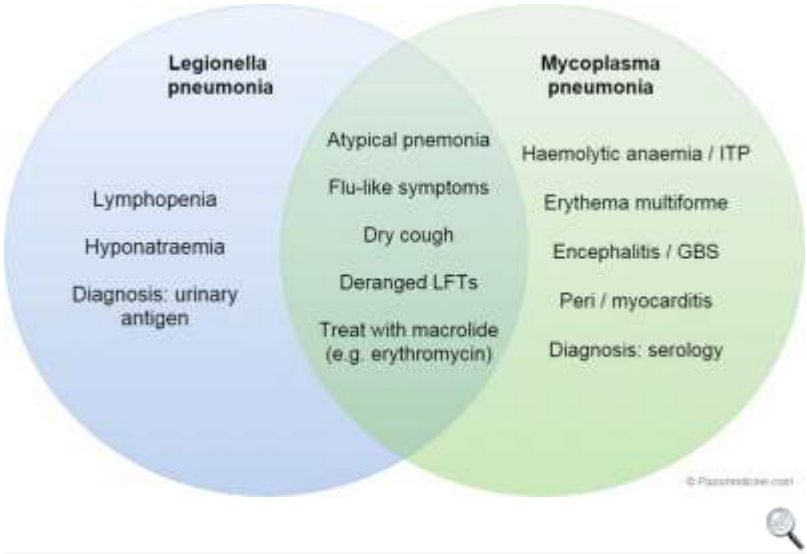
Investigations

- diagnosis is generally by Mycoplasma serology

- positive cold agglutination test → peripheral blood smear may show red blood cell agglutination

Management

- doxycycline or a macrolide (e.g. erythromycin/clarithromycin)



Comparison of Legionella and Mycoplasma pneumonia

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

11 6

Erythema multiforme

Suggest link Report broken link

Media



Mycoplasma pneumoniae

Osmosis - YouTube 1 0

Report broken media

Score: **15.8%**

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A 24-year-old man attends the sexual health clinic complaining of urethral discharge and dysuria following an episode of unprotected intercourse. Examination of the external genitalia confirms purulent discharge but no other abnormalities. There is no previous history of sexually transmitted infection.

You undertake microscopy of the discharge which reveals many white blood cells but no organisms. A urine sample is sent for nucleic acid amplification tests (NAAT) for *Chlamydia trachomatis* and *Neisseria gonorrhoea*. You treat him empirically with a course of doxycycline for presumed chlamydial infection.

He returns for review a week later. You note both NAAT results to be negative. However, the patient tells you his symptoms have fully resolved.

What is the most likely explanation?

- *Mycoplasma genitalium* infection
- False negative NAAT for *Chlamydia trachomatis*
- False negative NAAT for *Neisseria gonorrhoea*
- Herpes simplex virus infection
- Urinary tract infection

Submit answer

Reference ranges

Score: **32%**

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A 24-year-old man attends the sexual health clinic complaining of urethral discharge and dysuria following an episode of unprotected intercourse. Examination of the external genitalia confirms purulent discharge but no other abnormalities. There is no previous history of sexually transmitted infection.

You undertake microscopy of the discharge which reveals many white blood cells but no organisms. A urine sample is sent for nucleic acid amplification tests (NAAT) for *Chlamydia trachomatis* and *Neisseria gonorrhoea*. You treat him empirically with a course of doxycycline for presumed chlamydial infection.

He returns for review a week later. You note both NAAT results to be negative. However, the patient tells you his symptoms have fully resolved.

What is the most likely explanation?

Mycoplasma genitalium infection	60%
False negative NAAT for *Chlamydia trachomatis*	27%
False negative NAAT for *Neisseria gonorrhoea*	8%
Herpes simplex virus infection	2%
Urinary tract infection	3%

Urethritis in a male, negative for *Gonorrhoea* and *Chlamydia* →?*Mycoplasma genitalium*

Important for me Less important

The correct answer is ***Mycoplasma genitalium* infection**. This has emerged as the leading cause of non-gonococcal urethritis (NGU). Urethritis is evidenced in this case by the symptoms of dysuria and discharge combined with the presence of white cells in the discharge. NGU is diagnosed when features of urethritis exist in the absence of a positive test for gonorrhoea. Other explanations include infection with *Chlamydia* or organisms such as *Ureaplasma urealyticum*.

False negative NAAT for *Chlamydia trachomatis* is incorrect. Although improvement with doxycycline would fit with *Chlamydia*, the sensitivity of NAATs reaches 97% in some studies, making this a less likely explanation.

False negative NAAT for *Neisseria gonorrhoea* is incorrect. The sensitivity of NAATs for

gonorrhoea reaches 98-99% in some studies. We would also not expect a symptomatic improvement with doxycycline.

Herpes simplex virus infection is incorrect. HSV most commonly presents with painful genital ulceration. Less commonly, patients may present with dysuria and discharge in the absence of visible ulceration. However, we are told this patient has had no previous STIs. The first episode of genital HSV is often severe and prolonged in the absence of anti-viral treatment and the spontaneous improvement in 7 days described here would be unusual. Subsequent recurrences are often less severe and more short-lived.

Urinary tract infection is incorrect. Urinary tract infection frequently causes dysuria but urethral discharge would be an unusual feature. It is also very unusual to see urinary tract infections in young males in the absence of a structural abnormality of the urinary tract or an iatrogenic cause such as catheterisation.

Discuss (1) Improve

Next question

Non-gonococcal urethritis

Non-gonococcal urethritis (NGU, sometimes referred to as non-specific urethritis) is a term used to describe the presence of urethritis when a gonococcal bacteria are not identifiable or the initial swab. A typical case would be a male who presented to a GUM clinic with a purulent urethral discharge and dysuria. A swab would be taken in clinic, microscopy performed which showed neutrophils but no Gram-negative diplococci (i.e. no evidence of gonorrhoea). Clearly, this patient requires immediate treatment prior to waiting for the Chlamydia test to come back and hence an initial diagnosis of NGU is made.

No cause is found in around half of cases. Possible causative organisms include:

- *Chlamydia trachomatis*
  - most common cause
- *Mycoplasma genitalium*
  - thought to cause more symptoms than *Chlamydia*
- less common causes
  - *Ureaplasma urealyticum*
  - *Trichomonas vaginalis*
  - *Escherichia coli*

Management

- contact tracing
- the BNF and British Association for Sexual Health and HIV (BASHH) both recommend either oral azithromycin or doxycycline

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Association for Sexual Health and HIV (BASHH) 4 2

2015 UK National Guideline on the management of non-gonococcal urethritis

Suggest link Report broken link

Score: **15.8%**

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Back to top

A 28-year-old woman presents to the genitourinary medicine clinic with four days of dysuria and vaginal discharge. She has no past medical history and is on no regular medications. She had unprotected sexual intercourse with a casual partner two weeks previously.

Speculum examination revealed cervicitis and clear vaginal discharge. Examination of the genitalia was otherwise unremarkable.

A nucleic acid amplification test demonstrates the culprit organism.

Based on the clinical history, the patient is prescribed doxycycline and makes a full recovery.

Which organism is likely to be responsible for the clinical presentation?

- *Trichomonas vaginalis*
- *Candida albicans*
- *Chlamydia trachomatis*
- *Gardnerella vaginalis*
- *Neisseria gonorrhoeae*

Submit answer

Reference ranges

Score: **32%**

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Speculum examination revealed cervicitis and clear vaginal discharge. Examination of the genitalia was otherwise unremarkable.

A nucleic acid amplification test demonstrates the culprit organism.

Based on the clinical history, the patient is prescribed doxycycline and makes a full recovery.

Which organism is likely to be responsible for the clinical presentation?

Trichomonas vaginalis	8%
Candida albicans	0%
Chlamydia trachomatis	83%
Gardnerella vaginalis	4%
Neisseria gonorrhoeae	5%

Chlamydia - treat with doxycycline

Important for me Less important

Chlamydia trachomatis is the correct answer. This sexually transmitted infection can either be asymptomatic or may present with dysuria, cervicitis and a clear vaginal discharge. It is diagnosed by a nucleic acid amplification test. The first line antibiotic is doxycycline.

Trichomonas vaginalis is incorrect. This can cause dysuria and cervicitis and is also diagnosed by a nucleic acid amplification test.. However, the discharge tends to be green-ish and the treatment is with metronidazole.

Candida albicans is incorrect. The clinical picture in this case tends to be a creamy, itchy discharge. It is a fungal infection and is treated with topical or oral anti-fungal agents.

Gardnerella vaginalis is incorrect. This is a typical cause of bacterial vaginosis. A 'fishy' odour is commonly described in this disorder and the treatment is with metronidazole.

Neisseria gonorrhoeae is incorrect. This shares many of the same symptoms as *Chlamydia trachomatis* and can also be diagnosed via a nucleic acid amplification test. However, the treatment is with ceftriaxone.

Discuss (1) | Improve

Next question >

Chlamydia

Chlamydia is the most prevalent sexually transmitted infection in the UK and is caused by *Chlamydia trachomatis*, an obligate intracellular pathogen. Approximately 1 in 10 young women in the UK have *Chlamydia*. The incubation period is around 7-21 days, although it should be remembered a large percentage of cases are asymptomatic

Features

- asymptomatic in around 70% of women and 50% of men
- women: cervicitis (discharge, bleeding), dysuria
- men: urethral discharge, dysuria

Potential complications

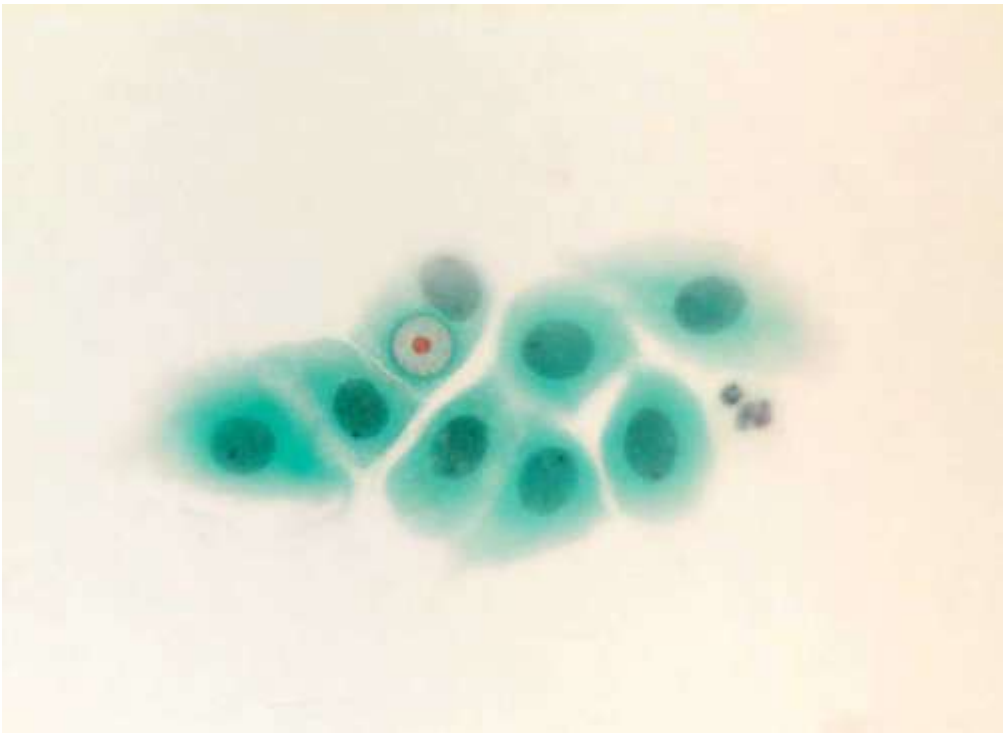
- epididymitis
- pelvic inflammatory disease
- endometritis
- increased incidence of ectopic pregnancies
- infertility
- reactive arthritis
- perihepatitis (Fitz-Hugh-Curtis syndrome)

Investigation

- traditional cell culture is no longer widely used
- nuclear acid amplification tests (NAATs) are now the investigation of choice
- urine (first void urine sample), vulvovaginal swab or cervical swab may be tested using the NAAT technique
- for women: the vulvovaginal swab is first-line
- for men: the urine test is first-line
- *Chlamydia*testing should be carried out two weeks after a possible exposure

Screening

- in England the National *Chlamydia* Screening Programme is open to all men and women aged 15-24 years
- the 2009 SIGN guidelines support this approach, suggesting screening all sexually active patients aged 15-24 years
- relies heavily on opportunistic testing



© Image used on license from PathoPic

Pap smear demonstrating infected endocervical cells. Red inclusion bodies are typical

Management

- doxycycline (7 day course) if first-line
  - this is now preferred to azithromycin due to concerns about *Mycoplasma genitalium*. This infection is often coexistant in patients with *Chlamydia* and there is evidence of rising levels of macrolide resistance, hence why doxycycline is preferred
  - if doxycycline is contraindicated / not tolerated then either azithromycin (1g od for one day, then 500mg od for two days) should be used
- if pregnant then azithromycin, erythromycin or amoxicillin may be used. The SIGN guidelines suggest azithromycin 1g stat is the drug of choice 'following discussion of the balance of benefits and risks with the patient'
- patients diagnosed with *Chlamydia* should be offered a choice of provider for initial partner notification - either trained practice nurses with support from GUM, or referral to GUM
- for men with urethral symptoms: all contacts since, and in the four weeks prior to, the onset of symptoms
- for women and asymptomatic men all partners from the last six months or the most recent sexual partner should be contacted
- contacts of confirmed *Chlamydia* cases should be offered treatment prior to the results of their investigations being known (treat then test)

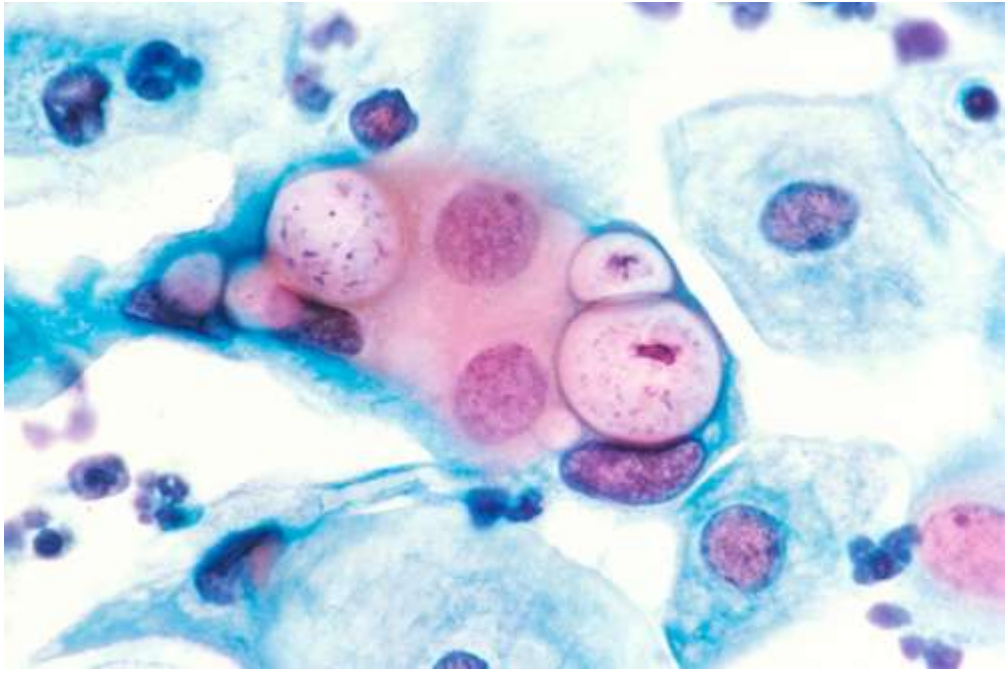


Image sourced from Wikipedia© Image used on license from PathoPic

Another Pap smear demonstrating infected endocervical cells. Stained with H&E

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Association for Sexual Health and HIV

12 11

2018 UK national guideline for the management of infection with Chlamydia trachomatis

Public Health England

13 11

Sexually transmitted infections and chlamydia screening in England, 2014

Clinical Knowledge Summaries

Chlamydia guidelines

18 8

Suggest link Report broken link

Score: **15.8%**

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A 28-year-old male presents to the emergency department complaining of an 8-day history of red eyes, fever, myalgia and headache. He has no significant past medical history and is on no regular medications. He has recently returned from a holiday with friends where they were kayaking and swimming in a freshwater lake. They had to abandon their trip earlier than expected as there was concerns about exposure to raw sewage in the lake.

His observations are heart rate 87 beats per minute, blood pressure 111/56 mmHg, respiratory rate 19/minute, oxygen saturation 96% on air and temperature 38.3ºC.

On examination, there is bilateral erythema of the conjunctiva. Cardiovascular and respiratory examinations are unremarkable. Abdominal examination reveals mild right upper quadrant tenderness and hepatomegaly. There is no photophobia or nuchal rigidity.

Urinalysis is unremarkable.

Plain radiography of the chest demonstrates clear lung fields.

Blood tests:

Hb	141 g/L	Male: (135-180) Female: (115 - 160)
Platelets	414 * 10^9/L	(150 - 400)
WBC	11.8 * 10^9/L	(4.0 - 11.0)
Na^+	141 mmol/L	(135 - 145)
K^+	4.1 mmol/L	(3.5 - 5.0)
Urea	9.2 mmol/L	(2.0 - 7.0)
Creatinine	141 µmol/L	(55 - 120)
CRP	68 mg/L	(< 5)
Bilirubin	38 µmol/L	(3 - 17)
ALP	167 u/L	(30 - 100)
ALT	190 u/L	(3 - 40)
Î³GT	111 u/L	(8 - 60)
Albumin	34 g/L	(35 - 50)

Given the clinical context, what is the most appropriate investigation to make the diagnosis?

- Blood culture
- Urine polymerase chain reaction (PCR)
- Serology
- Urinary antigens
- Urine culture

Submit answer

Reference ranges

Score: **32%**

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CRP	68 mg/L	(< 5)
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ALP	167 u/L	(30 - 100)
ALT	190 u/L	(3 - 40)
Î³GT	111 u/L	(8 - 60)
Albumin	34 g/L	(35 - 50)

Given the clinical context, what is the most appropriate investigation to make the diagnosis?

Blood culture	5%
Urine polymerase chain reaction (PCR)	14%
Serology	65%
Urinary antigens	15%
Urine culture	1%

Serology is the most commonly used test for the diagnosis of leptospirosis, however, antibodies may not be present until after 7 days

Important for me Less important

Serological testing is the correct answer. The diagnosis is leptospirosis, as evidenced by a history of fever, myalgia, headache and subconjunctival haemorrhage in association with hepatorenal failure in a patient with a potential geographic exposure to infected urine (freshwater exposure). The most commonly used test to diagnose leptospirosis is serological testing for IgM antibodies to leptospirosis. The disadvantages with this method of investigation are false positives, the inability for the test to diagnose early infection and the fact that antibodies can persist in the sera for months after initial infection, making interpretation of timing of infection difficult in cases of diagnostic uncertainty. Antibodies are not usually present until the infection has been present for seven days. In this case, the patient has presented with eight days of symptoms and therefore antibody testing should be positive.

Blood culture is the wrong answer. Culture testing for leptospirosis can take up to three months and is therefore predominantly used for retrospective diagnosis.

Urine polymerase chain reaction (PCR) testing is incorrect. While this technique is effective in identifying leptospira infection, it is not as commonly used as serological testing as its use is precluded by cost and technical factors in non-reference laboratories.

Urinary antigen testing is incorrect. This is used to diagnose pneumococcal and legionella pneumonia infections. The clinical history is not suggestive of a pneumonia.

Urine culture is incorrect. The use of this method of testing is precluded by the fact that cultures may take 8-12 weeks to form.

Discuss (4) Improve

Next question

Leptospirosis

Leptospirosis is caused by the spirochaete *Leptospira interrogans* (serogroup *L. icterohaemorrhagiae*), classically being spread by contact with infected rat urine.

Epidemiology

- leptospirosis is commonly seen in questions referring to sewage workers, farmers, vets or people who work in an abattoir
- however, on an international level, leptospirosis is far more common in the tropics so should be considered in the returning traveller

Weil's disease should always be considered in high-risk patients with hepatorenal failure

Features

- the early phase is due to bacteraemia and lasts around a week
  - may be mild or subclinical
  - fever
  - flu-like symptoms
  - subconjunctival suffusion (redness)/haemorrhage
- second immune phase may lead to more severe disease (Weil's disease)
  - acute kidney injury (seen in 50% of patients)
  - hepatitis: jaundice, hepatomegaly
  - aseptic meningitis

Investigation

- **serology**: antibodies to *Leptospira* develop after about 7 days
- PCR
- culture
  - growth may take several weeks so limits usefulness in diagnosis
  - blood and CSF samples are generally positive for the first 10 days
  - urine cultures become positive during the second week of illness

Management

- high-dose benzylpenicillin or doxycycline

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Quarterly Journal of Medicine 12 4

Leptospirosis review

Suggest link Report broken link

Media



Leptospira

Osmosis - YouTube 7 0

Report broken media

Score: **15.8%**

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A 34-year-old man is referred to the medical admissions unit with joint pain and swelling in his hands and feet. One month prior to his attendance, he had developed an illness while travelling in Colombia, which was characterized by fever, headache, rash and debilitating polyarthralgia. His treating physicians noted mild hepatomegaly at the time. He recovered well from this episode without intervention, except for his ongoing musculoskeletal symptoms.

Examination reveals a small joint polyarthritis of the hands and feet. Abdominal examination was unremarkable.

Serological testing is positive for an RNA virus.

What is the likely diagnosis?

- Chikungunya virus
- Dengue
- Epstein Barr Virus (EBV)
- Parvovirus
- Zika virus

Submit answer

Reference ranges

Score: **32%**

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Examination reveals a small joint polyarthritis of the hands and feet. Abdominal examination was unremarkable.

Serological testing is positive for an RNA virus.

What is the likely diagnosis?

Chikungunya virus	76%
Dengue	10%
Epstein Barr Virus (EBV)	3%
Parvovirus	3%
Zika virus	8%

Chikungunya can present with debilitating joint pain

Important for me Less important

Chikungunya is the correct answer. This is an RNA alpha-virus characterized by sudden onset fever accompanied by skin rashes and joint pain followed by persistent rheumatic symptoms. In contrast to the other diagnoses, which can all cause headache, fever and rash, the polyarthralgia is said to be far more debilitating and it is most associated with the development of persistent musculoskeletal symptoms. Furthermore, hepatomegaly can be a feature of this condition but this is not seen in Zika or dengue. While hepatomegaly is a feature of EBV, this is a DNA virus and is less likely to cause persistent musculoskeletal symptoms.

Zika virus is characterized by a pruritic maculopapular rash accompanied by other symptoms such as fever, headache, conjunctivitis, myalgia and arthralgia. It is an RNA virus but is much less likely to cause persistent musculoskeletal symptoms.

Dengue is incorrect. This is another RNA arbovirus that causes fever, rash and arthralgia in

addition to other features such as thrombocytopenia. However, hepatomegaly is not a feature and it is not associated with the development of persistent arthritis.

Epstein Barr Virus (EBV) is incorrect. This is a DNA virus. Musculoskeletal manifestations are less prominent.

Parvovirus is incorrect. This can cause rash, hepatomegaly and polyarthritis but it is a DNA virus.

Discuss (5) Improve

Next question ›

Chikungunya

Alphavirus disease caused by infected mosquitoes. Areas affected are Africa, Asia and Indian subcontinent but in recent years there has been seen in a few cases in Southern Europe. Tanzania had the first reported case.

Symptoms: Prominent symptoms are severe joint pain and abrupt onset of high fever. Other symptoms include general flu-like illness of muscle ache, headache, and fatigue. The disease shares its symptoms with dengue but tends to have more joint pain which can be debilitating. A rash may develop as with other viral illness and swelling of the joints in not uncommon.

Treatment: Relief of symptoms. No specific treatment.

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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Back to top

A 38-year-old farmer is admitted to the acute medical unit with a 2-week history of fever and dry cough. He describes feeling increasingly more lethargic with generalised body aches and headaches. He has no past medical history of note and takes no regular medications.

On auscultation, there are scattered crackles. His cardiovascular examination is unremarkable. There is mild tenderness in the right upper quadrant, but his abdomen remains soft with no palpable organomegaly.

Laboratory tests:

Hb	139 g/L	(135-180)
Platelets	499 * 10^9/L	(150 - 400)
WBC	16.1 * 10^9/L	(4.0 - 11.0)
Na^+	144 mmol/L	(135 - 145)
K^+	4.6 mmol/L	(3.5 - 5.0)
Urea	6.1 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
Bilirubin	15 µmol/L	(3 - 17)
ALP	80 u/L	(30 - 100)
ALT	120 u/L	(3 - 40)
CRP	199 mg/L	(< 5)

What is the most likely causative organism for this patient's presentation?

- *Brucella melitensis*
- *Coxiella burnetii*
- *Mycoplasma pneumoniae*
- *Staphylococcus aureus*
- *Streptococcus pneumoniae*

Submit answer

Reference ranges

Score: **32%**

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K^+	4.6 mmol/L	(3.5 - 5.0)
Urea	6.1 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
Bilirubin	15 µmol/L	(3 - 17)
ALP	80 u/L	(30 - 100)
ALT	120 u/L	(3 - 40)
CRP	199 mg/L	(< 5)

What is the most likely causative organism for this patient's presentation?

- *Brucella melitensis* 19%
- *Coxiella burnetii* 63%
- *Mycoplasma pneumoniae* 15%
- *Staphylococcus aureus* 1%
- *Streptococcus pneumoniae* 2%

Coxiella burnetii is a cause of atypical pneumonia and Q fever. It is usually transmitted by infected cattle or sheep

This patient has an infection with ***Coxiella burnetii*** a zoonotic disease more commonly seen in those who work with animals such as farmers and veterinarians. Infection gives rise to Q fever that can present with a variety of symptoms ranging from fever, malaise and myalgia to atypical pneumonia or infective endocarditis. Auscultatory crackles and a dry cough with fever and fatigue are suggestive of atypical pneumonia. Furthermore, *Coxiella burnetii* can cause transaminitis as demonstrated by this patient's deranged liver function tests.

Brucella melitensis is another zoonotic disease secondary to close contact with infected animals or from ingestion of unpasteurised milk or undercooked meat. It is more common in the Middle East, farmers, vets, and abattoir workers. Symptoms include fever and malaise, as seen here. However, features of pneumonia are not common in Brucellosis making it a less likely diagnosis.

A ***Mycoplasma pneumoniae*** infection commonly presents with a preceding phase of flu-like symptoms before the onset of respiratory symptoms including shortness of breath and productive cough. Extra-pulmonary symptoms are common in patients with *Mycoplasma pneumoniae* and include erythema multiforme and haemolytic anaemia. The absence of these extra-pulmonary symptoms and the occupational history as a farmer make the diagnosis of Q fever more likely.

Staphylococcus aureus is a common cause of pneumonia in patients following infection with the influenza virus. Although this patient gives an initial history of flu-like symptoms, it does not explain his transaminitis and suggests an alternative diagnosis is more likely.

Streptococcus pneumoniae is the most common bacterial cause of community-acquired pneumonia. However, it does not explain this patient's transaminitis or prodrome of fever and myalgia. Furthermore, an occupational history as a farmer makes alternative zoonotic diseases, such as Q fever, more likely.

Discuss (3) Improve

Next question >

Q fever

Q fever is caused by *Coxiella burnetii*, a rickettsia. The source of infection is typically an abattoir, cattle/sheep or it may be inhaled from infected dust

Features

- typically prodrome: fever, malaise
- causes pyrexia of unknown origin
- transaminitis
- atypical pneumonia
- endocarditis (culture-negative)

Management

- doxycycline

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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Back to top

A 49-year-old man is admitted to the acute medical unit with a 2-week history of pain and difficulty when swallowing. He reports having lost approximately 2-3kg in weight from reduced oral intake. His past medical history includes previous hospital admissions for lower respiratory tract infections and lower limb deep vein thrombosis.

On examination, he appears cachectic. There are multiple, well-defined white plaques over the oral mucosa. His abdomen is soft and non-tender with no palpable masses.

CD4 count	< 100 cells/mm³

What is the most appropriate management for this patient?

- Aciclovir
- Caspofungin
- Co-amoxiclav and metronidazole
- Fluconazole
- Nystatin and chlorhexidine mouthwash

Submit answer

Reference ranges

Score: **32%**

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On examination, he appears cachectic. There are multiple, well-defined white plaques over the oral mucosa. His abdomen is soft and non-tender with no palpable masses.

CD4 count	< 100 cells/mm³

What is the most appropriate management for this patient?

Aciclovir	3%
Caspofungin	3%
Co-amoxiclav and metronidazole	1%
Fluconazole	78%
Nystatin and chlorhexidine mouthwash	14%

Candidiasis in immunocompromised patients should be treated using high dose fluconazole

Important for me Less important

This patient has a diagnosis of oesophageal candidiasis given the classical history of painful swallowing and visible white mucosal plaques on examination. It is particularly more common in immunocompromised individuals (e.g. such as patients with HIV). The treatment of choice for treating candidiasis in immunocompromised patients is with high-dose **fluconazole** or itraconazole.

Aciclovir is used to treat viral infections, including herpes simplex and herpes zoster. Typically viral rashes such as herpes simplex present with ulceration or vesicles. The presence of a white mucosal coating in an immunocompromised patient is highly suggestive of candidiasis, for which antifungal medication is needed, rather than antivirals like aciclovir.

Caspofungin is not a first-line treatment for oesophageal candidiasis. It is reserved for cases of refractory disease that do not respond to initial treatment with fluconazole. As first-line therapy has not yet been initiated, it would be inappropriate to start the patient on more invasive

treatment such as caspofungin.

Co-amoxiclav and metronidazole would be indicated if there were concerns that this patient was suffering from a bacterial infection. However, the classical history of odynophagia and white mucosal coating in an immunocompromised patient makes candidiasis much more likely to be the diagnosis.

Nystatin and chlorhexidine mouthwash would normally form part of the initial management in immunocompetent patients. However, this patient has a CD4 count of < 100 cells/mm^3 suggesting significant immunocompromise. Therefore, stronger treatment such as high-dose fluconazole is needed.

Discuss (1) | Improve

Next question ›

HIV: oesophageal candidiasis

Oesophageal candidiasis is the most common cause of oesophagitis in patients with HIV. It is generally seen in patients with a CD4 count of less than 100. Typical symptoms include dysphagia and odynophagia. Fluconazole and itraconazole are first-line treatments.

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media



Candidal infections

Osmosis - YouTube 2 1

Report broken media

Score: **15.8%**

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A 55 year old man attended his General Practitioner to complain of on-going fatigue and lethargy over recent months. He reported experiencing recurrent minor infections meaning that he had rarely been feeling well for any significant length of time. His GP records supported this story with the patient having been prescribed courses of antibiotics for a leg cellulitis, suppurative otitis media and sinusitis (twice) over the past 9 months. The patient reported a good appetite and no recent weight loss, fevers or night sweats. He denied any symptoms of arthritis, skin rashes, photophobia or dry eyes. There were no significant gastrointestinal or genitourinary symptoms.

Past medical history included obesity, hypertension, impaired fasting glucose tolerance and osteoarthritis of the knees. Drug therapy included ramipril 2.5 mg daily, bendroflumethiazide 2.5 mg daily and paracetamol as required. The patient was divorced with two grown up children. He was an ex-smoker who rarely drank alcohol. The patient had recently retired having previously worked as a salesperson for luxury yachts, an occupation that had involved extensive travel around the world.

On examination, the patient was significantly overweight (BMI 36 kg / m^2). Abdominal examination did not demonstrate any jaundice or signs of chronic liver disease and no lymphadenopathy. Cardiovascular, respiratory and musculoskeletal examination was unremarkable. Given the patients concerns, his General Practitioner arranged some basic blood tests.

Haemoglobin	16.0 g / dL
Mean cell volume	85 fL
White cell count	5.2 * 10^9/l
Neutrophils	4.2 * 10^9/l
Lymphocytes	0.6 * 10^9/l
Monocytes	0.1 * 10^9/l
Eosinophils	0.1 * 10^9/l
Basophils	0.2 * 10^9/l
Platelets	358 * 10^9/l
Urea	6.8 mmol / L
Creatinine	110 micromol / L
Sodium	141 mmol / L
Potassium	3.9 mmol / L
Albumin	34 g / L (reference 35-50)
Alkaline phosphatase	98 U / L (reference (35-100)
ALT	28 U / L (reference 3-36)

Bilirubin	21 micromol / L (reference < 26)
Total protein	85 g / L (reference 60-80)
B12	355 pmol / L (reference 74-516)
Folate	30 nmol / L (reference 7-36)
Serum immunoglobulins	Normal electrophoresis strip

Comparison with a routine blood test taken 6 months previously had shown a similar full blood count differential.

What is the most appropriate next line investigation?

- ○ Human immunodeficiency virus antibody testing ×
- ○ Anti-nuclear antibody ×
- ○ Rheumatoid factor ×
- ○ Epstein-Barr virus serology ×
- ○ Serum angiotensin-converting enzyme ×

Submit answer

Reference ranges

Score: **32%**

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Serum immunoglobulins	Normal electrophoresis strip

Comparison with a routine blood test taken 6 months previously had shown a similar full blood count differential.

What is the most appropriate next line investigation?

- Human immunodeficiency virus antibody testing — 82%
- Anti-nuclear antibody — 4%
- Rheumatoid factor — 1%
- Epstein-Barr virus serology — 9%
- Serum angiotensin-converting enzyme — 5%

The patient has a persistent lymphopenia, raised total protein and low albumin in the setting of fatigue and recurrent minor infections. These findings could all be explained by nascent HIV infection. The patients history of international travel may place him at increased risk of HIV infection and a full sexual history would be required.

The other investigations listed would assess for other possible causes of isolated lymphopenia, however there is no clinical evidence of connective tissue disease, EBV infection or sarcoidosis to

Brass D, Mckay P, Scott F. Investigation an incidental finding of lymphopenia. BMJ 2014;348:g1721.

Discuss (7) Improve

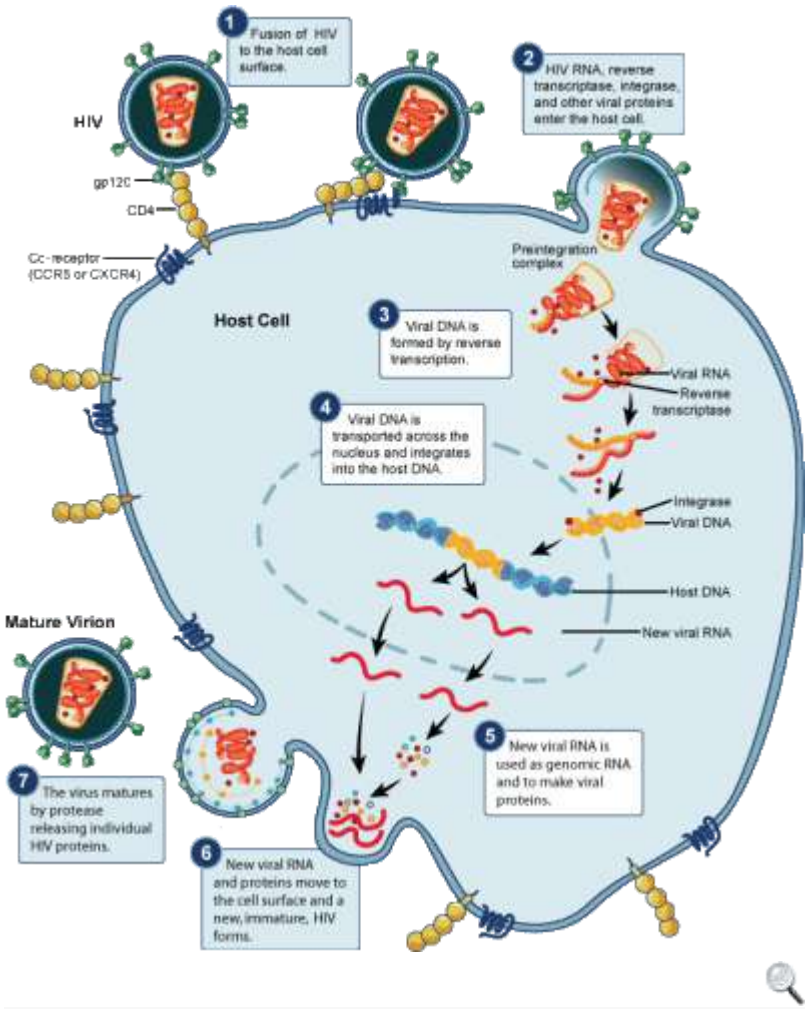
Next question >

HIV: seroconversion ★

HIV seroconversion is symptomatic in 60-80% of patients and typically presents as a glandular fever-type illness. Increased symptomatic severity is associated with poorer long-term prognosis. It typically occurs 3-12 weeks after infection

Features

- sore throat
- lymphadenopathy
- malaise, myalgia, arthralgia
- diarrhoea
- maculopapular rash
- mouth ulcers
- rarely meningoencephalitis



An illustration model of the HIV Replication Cycle. Each step of the cycle is numbered and concisely described. Credit: NIAID

123

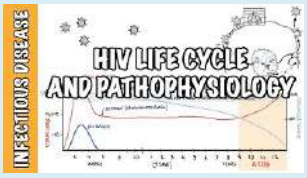
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Human immunodeficiency virus (HIV) - life cycle, pathophysiology, investigations, diagnosis

Armando Hasudungan - YouTube

2 0



HIV

Osmosis - YouTube

4 2

Report broken media

Score: **15.8%**

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Back to top

A 28-year-old man who has recently immigrated from Nigeria presents with a penile ulcer. He reports that it initially started as a small lump but then later progressed to a painful ulcer.

On examination, there is a 7mm diameter tender single ulcer with an undermined ragged edge just proximal to the glans of the penis. Examination of the testes and anal region is unremarkable. There is tender inguinal lymphadenopathy.

What is the most likely diagnosis?

- Syphilis
- Herpes simplex virus
- Granuloma inguinale
- Lymphogranuloma venereum
- Chancroid

Submit answer

Reference ranges

Score: **32%**

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On examination, there is a 7mm diameter tender single ulcer with an undermined ragged edge just proximal to the glans of the penis. Examination of the testes and anal region is unremarkable. There is tender inguinal lymphadenopathy.

What is the most likely diagnosis?

Syphilis	4%
Herpes simplex virus	6%
Granuloma inguinale	9%
Lymphogranuloma venereum	32%
Chancroid	**50%**

Chancroid is a bacterial sexually transmitted infection characterised by painful sores on the genitalia. It is caused by the gram-negative coccobacillus *Haemophilus ducreyi*. It is a disease found primarily in developing countries.

H. ducreyi enters the skin through microabrasions incurred during sexual intercourse. A local tissue reaction leads to the development of an erythematous papule, which progresses to a pustule in 4-7 days. It then undergoes central necrosis to ulcerate.

The ulcer can range in size from 3 to 50 mm across. It is painful and has sharply defined undermined borders

Other features include:

- Irregular or ragged borders
- A base that is covered with a grey or yellowish-gray material
- A base that bleeds easily if traumatised or scraped
- Painful lymphadenopathy in 30 to 60% of patients.
- Dysuria and dyspareunia in females

About half of infected men have only a single ulcer. Women frequently have four or more ulcers, with fewer symptoms.

Sexually transmitted infections: ulcers

Genital herpes is most often caused by the herpes simplex virus (HSV) type 2 (cold sores are usually due to HSV type 1). Primary attacks are often severe and associated with fever whilst subsequent attacks are generally less severe and localised to one site. There is typically multiple painful ulcers.

Syphilis is a sexually transmitted infection caused by the spirochaete *Treponema pallidum*. Infection is characterised by primary, secondary and tertiary stages. A painless ulcer (chancre) is seen in the primary stage. The incubation period= 9-90 days.

Chancroid is a tropical disease caused by *Haemophilus ducreyi*. It causes painful genital ulcers associated with unilateral, painful inguinal lymph node enlargement. The ulcers typically have a sharply defined, ragged, undermined border.

Lymphogranuloma venereum (LGV) is caused by *Chlamydia trachomatis*. Typically infection comprises of three stages

- stage 1: small painless pustule which later forms an ulcer
- stage 2: painful inguinal lymphadenopathy
- stage 3: proctocolitis

LGV is treated using doxycycline.

Other causes of genital ulcers

- Behcet's disease
- carcinoma
- granuloma inguinale: *Klebsiella granulomatis**

*previously called *Calymmatobacterium granulomatis*

Textbooks

High-yield textbook

Extended textbook

Links

British Association for Sexual Health and HIV

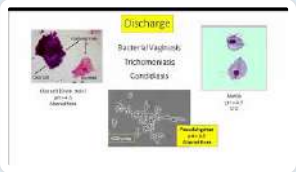
9 3

2018 Guideline for the management of Donovanosis (Granuloma inguinale)

Suggest link

Report broken link

Media



Genital Infections for USMLE Step One

12DaysinMarch - YouTube 6 0

Report broken media

Score: **15.8%**

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Back to top

The Medical Emergency Team (MET) is summoned to the Surgical Unit to assist with the management of an acutely unwell patient.

The patient is a 21-year-old male who underwent open surgery for perforated appendicitis 3 days ago. The Surgical Registrar informs you that faecal contamination of the abdomen was noted during the operation and that a peritoneal washout was performed.

24 hours later, the patient began to complain of worsening abdominal pain. He became febrile in the early hours of the morning and blood cultures were taken. Since then, he has become progressively more unwell. He was taken down for an urgent abdominal ultrasound mid-afternoon, but the nurses were so concerned about his condition when he arrived back on the ward that a MET call was put out.

On examination, the patient is responsive to voice. He is febrile at 38.9ºC, his pulse is 131bpm and his blood pressure is 72/53mmHg. His peripheries are warm and clammy. Palpation of the abdomen reveals localised tenderness and guarding in the right iliac fossa. The surgical wound appears clean with minimal surrounding erythema.

As you prepare to place a large bore IV cannula the Surgical FY1 passes you some results that have recently been phoned through:

Abdominal ultrasound	Anechoic fluid collection in the right iliac fossa
Blood culture	Gram-positive cocci both bottles - further information to follow

Which of the following organisms is most likely to be isolated from the blood culture?

- Staphylococcus epidermidis
- Escherichia coli
- Streptococcus pyogenes
- Enteroccocus faecalis
- Staphylococcus aureus

Submit answer

Reference ranges

Score: **32%**

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As you prepare to place a large bore IV cannula the Surgical FY1 passes you some results that have recently been phoned through:

Abdominal ultrasound	Anechoic fluid collection in the right iliac fossa
Blood culture	Gram-positive cocci both bottles - further information to follow

Which of the following organisms is most likely to be isolated from the blood culture?

Staphylococcus epidermidis	16%
Escherichia coli	5%
Streptococcus pyogenes	10%
Enteroccocus faecalis	53%
Staphylococcus aureus	16%

Although on first glance this may appear to be a 'surgical case', it is important to remember that MET or 'crash' teams are called to attend a variety of patients, some of whom may be admitted under the care of other specialties. On-call medical doctors need to be able to apply their

knowledge to a range of situations, particularly when they fall outwith the remit of their day-to-day practice.

A significant amount of surgical information is presented in this vignette, however, the underlying diagnosis is given to the candidate and the question is actually designed to test the candidate's knowledge of pathogenic bacteria and the illnesses they cause.

Strep. pyogenes is a common cause of pharyngitis and soft tissue infections, occasionally giving rise to septicaemia. It would be unlikely to cause illness in the case described above.

Staph. aureus and Staph. epidermidis are commensal organisms that commonly cause septicaemia in critically ill patients, often secondary to the colonisation of indwelling lines. This vignette makes no mention of any such devices and we are told that the surgical wound is clean, making primary staphylococcal wound infection unlikely.

Escherichia coli is a gram-negative bacillus and is, therefore, inconsistent with the blood culture results described.

Enterococcus faecalis is a group D streptococcus. It is a gut commensal and a well-known cause of intra-abdominal infections. In the case described, it is likely that faecal contamination of the abdomen lead to the formation of an abscess and consequent Enterococcus bacteraemia.

Discuss (8) | Improve

Next question >

Streptococci

Streptococci are gram-positive cocci. They may be divided into alpha and beta haemolytic types

Alpha haemolytic streptococci (partial haemolysis)

The most important alpha haemolytic *Streptococcus* is *Streptococcus pneumoniae* (pneumococcus). Pneumococcus is a common cause of pneumonia, meningitis and otitis media. Another clinical example is *Streptococcus viridans*

Beta haemolytic streptococci (complete haemolysis)

These can be subdivided into groups A-H. Only groups A, B & D are important in humans.

Group A

- most important organism is *Streptococcus pyogenes*

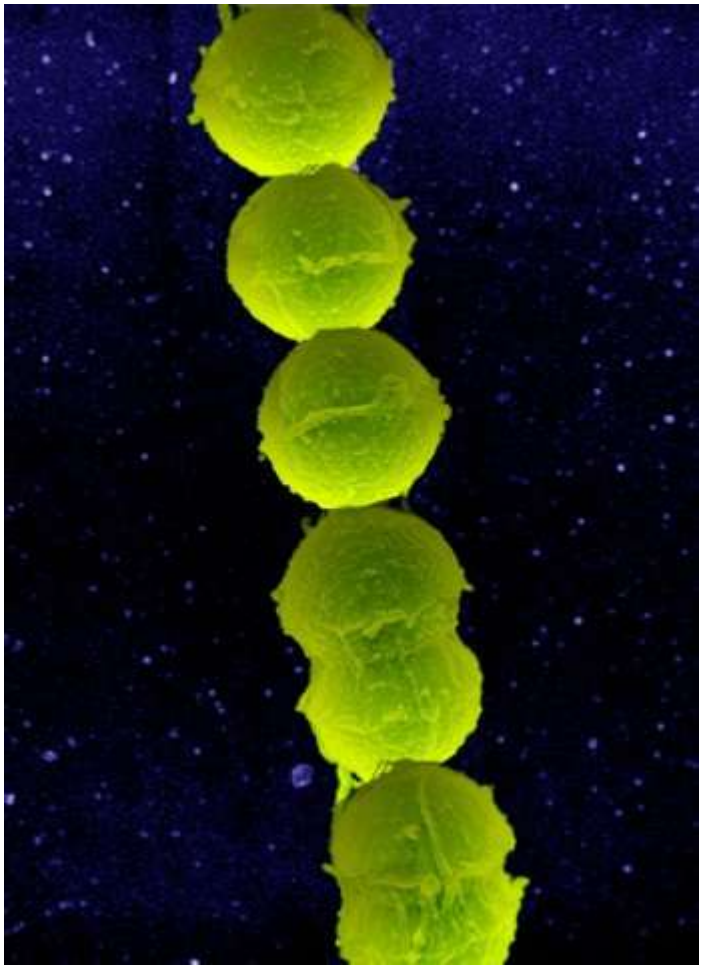
- responsible for erysipelas, impetigo, cellulitis, type 2 necrotizing fasciitis and pharyngitis/tonsillitis
- immunological reactions can cause rheumatic fever or post-streptococcal glomerulonephritis
- erythrogenic toxins cause scarlet fever

Group B

- *Streptococcus agalactiae* may lead to neonatal meningitis and septicaemia

Group D

- *Enterococcus*



Group B streptococcus bacteria. Credit: NIAID

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

3 1

Erysipelas

Suggest link Report broken link

Media



Streptococcus agalactiae

Osmosis - YouTube 1 0



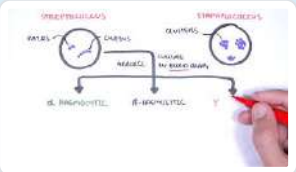
Streptococcus pneumoniae

Osmosis - YouTube 0 0



Enterococcus

Osmosis - YouTube 0 0



Microbiology - Streptococcus species

Armando Hasudungan - YouTube 1 1

Report broken media

Score: **15.8%**

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A 32 yr old Indian lady presented at the clinic with history of a gross swelling on her right leg from more than four years with associated fever and pain in her groin. Swelling was not reversible at night. On examination, the whole leg was oedematous ,bluish in colour, deep skin fold at junction of middle and lower one third, non discharging sinus above the fold anteriorly. There were no nodules or warts on her limbs.

Hb	144g/l (130 - 180)
Platelets	310 * 10^9/l (150 - 400)
WBC	9.2 * 10^9/l (4.0 - 11.0)
Neutrophil	3.5 * 10^9/l (1.5 - 7.0)
Lymphocyte	2.1 * 10^9/l (1.5 - 4.0
Eosinophil	2.4 * 10^9/l (0.04 - 0.4)
Monocyte	1.1 * 10^9/l (0<0.8)

What is the drug of choice for the most likely diagnosis for the scenario above?

- Praziquantel
- Niclosamide
- Ivermectin
- Diethylcarbamazine
- Metronidazole

Submit answer

Reference ranges

Score: **32%**

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Eosinophil	2.4 * 10^9/l (0.04 - 0.4)
Monocyte	1.1 * 10^9/l (0<0.8)

What is the drug of choice for the most likely diagnosis for the scenario above?

Praziquantel	15%
Niclosamide	3%
Ivermectin	27%
Diethylcarbamazine	52%
Metronidazole	3%

Diethylcarbamazine is the drug of choice for lymphatic filariasis

Important for me Less important

AI

Filariasis is a disease group caused by filariae that affects humans and animals. In lymphatic filariasis, repeated episodes of inflammation and lymphedema lead to lymphatic damage, chronic swelling, and elephantiasis of the legs. Eosinophilia is a feature of filariasis. Diethylcarbamazine is the drug of choice for lymphatic filariasis.

In contrast, Praziquantel is used to treat infections caused by Schistosoma worms, which enter the

body through skin that has come into contact with contaminated water. Symptoms include abdominal pain, diarrhea, bloody stool, or blood in the urine. Niclosamide is an antihelminthic that is active against tapeworm infestation which is transmitted mainly by eating infected raw or uncooked fish or beef and usually have intestinal discomfort, diarrhea, vomiting, weakness, and weight loss. Metronidazole is useful in treating infective diarrhoea with susceptible organisms.

Discuss (6) Improve

Next question

Wuchereria bancrofti

- Parasitic filarial nematode
- Accounts for 90% of cases of filariasis
- Usually diagnosed by blood smears
- Usually transmitted by mosquitos
- Treatment is with diethylcarbamazine

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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Randy is a 45-year-old male presenting with a new fever. He works as a farmer and noted fevers up to 39.6ºC over the past 72 hours with a dry cough. He has also been struggling to manage his sheep due to the ongoing headache and worsening muscle aches that have accompanied the fever. The headache was significant but was not associated with any neck stiffness or photophobia.

Randy does not note any other symptoms. He has not been in contact with anyone unwell and he has not been travelling out of the country recently.

Physical examination was otherwise unremarkable with only mild crackles heard on auscultation on his chest. No insect bites were noted. No lymphadenopathy was palpable.

Blood tests are as follow:

Hb	150 g/L	Male: (135-180) Female: (115 - 160)
Platelets	140 * 10^9/L	(150 - 400)
WBC	6.0 * 10^9/L	(4.0 - 11.0)
Na^+	140 mmol/L	(135 - 145)
K^+	3.9 mmol/L	(3.5 - 5.0)
Urea	6.0 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
Bilirubin	13 µmol/L	(3 - 17)
ALP	90 u/L	(30 - 100)
ALT	110 u/L	(3 - 40)
AST	100 u/L	(5-40)
Albumin	37 g/L	(35 - 50)

What would be the likely organism causing the likely diagnosis?

- *Rickettsia prowazekii*
- *Coxiella burnetti*
- *Francisella tularensis*
- *Legionella spp.*

○ *Bartonella henselae* ×

Submit answer

Reference ranges

Score: **32%**

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Urea	6.0 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
Bilirubin	13 µmol/L	(3 - 17)
ALP	90 u/L	(30 - 100)
ALT	110 u/L	(3 - 40)
AST	100 u/L	(5-40)
Albumin	37 g/L	(35 - 50)

What would be the likely organism causing the likely diagnosis?

Rickettsia prowazekii	7%
Coxiella burnetti	75%
Francisella tularensis	7%
Legionella spp.	3%

Bartonella henselae 8%

Farmer, fever, transaminitis ?Q fever

Important for me Less important

Given that the patient is a farmer, this exposes him to zoonotic infections. The presence of fevers and associated transaminitis raises the suspicion for Q fever, caused by *Coxiella burnetti*. Its natural reservoir exists within cattle and sheep. It typically presents with fevers, headaches, fatigue and muscle aches. Diagnosis of this is often made by serological testing and is treated with doxycycline. Chronic Q-fever is possible and can present with endocarditis.

Francisella tularensis causes tularemia. This is less likely given the lack of risk of exposure to its natural vector, rabbits or deer. Also, this will tend to present with unilateral lymphadenopathy with associated skin ulcers at the site of inoculation.

Legionella spp. causes Legionnaires disease which presents with fevers and headaches. However, this is less likely given the lack of risk factors of exposure including recent repairs to water supplies and exposure to a known Legionnaires case. Also, this tends to cause a productive cough rather than a dry cough.

Bartonella henselae causes cat-scratch disease and this is not likely given the absence of lymphadenopathy or rash at the site of inoculation. Besides, there is no mention that he has been exposed to a cat.

Rickettsia prowazekii causes epidemic typhus. This typically occurs in overcrowded areas as the disease is spread by body lice. This is also typically associated with a generalized rash beginning at the trunk before spreading to the extremities (sparing of face, palms and soles).

Discuss (1) Improve

Next question >

Q fever

Q fever is caused by *Coxiella burnetii*, a rickettsia. The source of infection is typically an abattoir, cattle/sheep or it may be inhaled from infected dust

Features

- typically prodrome: fever, malaise
- causes pyrexia of unknown origin
- transaminitis
- atypical pneumonia

- endocarditis (culture-negative)

Management
- doxycycline

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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A 23-year-old female presents to her general practitioner complaining of a two-day history of rash at the back of her knee. She has a past medical history of eczema and asthma and is on regular Symbicort and emollients. She is a non-smoker and non-drinker and has recently returned from a camping holiday where she had been doing some hiking through a forested area.

On examination, there is a bulls-eye lesion just above her right popliteal fossa. She is otherwise systemically well and apyrexial.

What is the most appropriate initial management?

- Oral doxycycline
- Oral flucloxacillin
- Serological testing for *Borrelia* antibodies
- Skin biopsy
- Topical steroid

Submit answer

Reference ranges

Score: **32%**

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On examination, there is a bulls-eye lesion just above her right popliteal fossa. She is otherwise systemically well and apyrexial.

What is the most appropriate initial management?

Oral doxycycline	89%
Oral flucloxacillin	1%
Serological testing for *Borrelia* antibodies	9%
Skin biopsy	1%
Topical steroid	1%

Lyme disease can be diagnosed clinically if erythema migrans is present → give antibiotics

Important for me Less important

Oral doxycycline is correct. This patient has a clinical diagnosis of Lyme disease and therefore treatment should be started as soon as possible. Doxycycline is the first-line antibiotic choice. NICE recommend a course length of 21 days. Second-line options include amoxicillin and azithromycin.

Serological testing for *Borrelia* antibodies is incorrect. NICE states that Lyme disease can be diagnosed clinically if erythema migrans is present. Given the typical appearance of the rash and the suggestive clinical history of a recent visit to a forest, we can make a clinical diagnosis in this case. This patient should receive antibiotics in the first instance and we do not need to wait for laboratory results.

Oral flucloxacillin is incorrect. This would be an appropriate choice for cellulitis but this is not the correct diagnosis.

Skin biopsy is incorrect. There is no uncertainty regarding diagnosis.

Topical steroid is the wrong answer. This would be a treatment for a flare of eczema but the description of the rash in association with the clinical history does not fit this description.

Discuss (1) | Improve

Next question >

Lyme disease ★

Lyme disease is caused by the spirochaete *Borrelia burgdorferi* and is spread by ticks.

Early features (within 30 days)

- erythema migrans
  - 'bulls-eye' rash is typically at the site of the tick bite
  - typically develops 1-4 weeks after the initial bite but may present sooner
  - usually painless, more than 5 cm in diameter and slowlly increases in size
  - present in around 80% of patients.
- systemic features
  - headache
  - lethargy
  - fever
  - arthralgia





Later features (after 30 days)

- cardiovascular
  - heart block
  - peri/myocarditis
- neurological
  - facial nerve palsy
  - radicular pain
  - meningitis



Investigation

- NICE recommend that Lyme disease can be diagnosed clinically if erythema migrans is present
  - erythema migrans is therefore an indication to start antibiotics
- enzyme-linked immunosorbent assay (ELISA) antibodies to *Borrelia burgdorferi* are the first-line test
  - if negative and Lyme disease is still suspected in people tested within 4 weeks from symptom onset, repeat the ELISA 4-6 weeks after the first ELISA test. If still suspected in people who have had symptoms for 12 weeks or more then an immunoblot test should be done
  - if positive or equivocal then an immunoblot test for Lyme disease should be done

Management of asymptomatic tick bites

- tick bites can be a relatively common presentation to GP practices, and can cause significant anxiety
- if the tick is still present, the best way to remove it is using fine-tipped tweezers, grasping the tick as close to the skin as possible and pulling upwards firmly. The area should be washed following.
- NICE guidance does not recommend routine antibiotic treatment to patients who've suffered a tick bite

Management of suspected/confirmed Lyme disease

- doxycycline if early disease. Amoxicillin is an alternative if doxycycline is contraindicated (e.g. pregnancy)
  - people with erythema migrans should be commenced on antibiotic without the need for further tests
- ceftriaxone if disseminated disease
- Jarisch-Herxheimer reaction is sometimes seen after initiating therapy: fever, rash, tachycardia after first dose of antibiotic (more commonly seen in syphilis, another spirochaetal disease)

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 11 13

Lyme disease: management

NICE 11 10

2018 Lyme disease guidelines

Dermnet NZ 12 5

Lyme disease

Suggest link Report broken link

Media



Lyme disease

Osmosis - YouTube 4 1

Report broken media

Score: **15.8%**

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Back to top

A 54-year-old man is brought into the emergency department with a 4-day history of fever, headache and reduced appetite. His partner brought him into the hospital today as he became more lethargic and drowsy today. He has a past medical history of coeliac disease and HIV.

On examination, his GCS is 12 (E2V4M6) with equal and reactive pupils. He has a temperature of 38.1ºC with a heart rate of 99 beats/min and a blood pressure of 135/80mmHg. There are no visible rashes.

A lumbar puncture is performed, the CSF results are shown below:

Opening pressure	30 cmH20	(10 - 20)
White cell count	50 x10^6/L mostly lymphocytes	(< 5)
Protein	3.5 g/L	(0.2 - 0.4)
Glucose	2 mmol/L	(2.8 - 4.2)

What is the most likely causative organism?

- *Cryptococcus neoformans*
- Herpes simplex virus
- *Listeria monocytes*
- *Neisseria meningitidis*
- *Streptococcus pneumoniae*

Submit answer

Reference ranges

Score: **32%**

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Protein	3.5 g/L	(0.2 - 0.4)
Glucose	2 mmol/L	(2.8 - 4.2)

What is the most likely causative organism?

Cryptococcus neoformans	68%
Herpes simplex virus	10%
Listeria monocytes	17%
Neisseria meningitidis	3%
Streptococcus pneumoniae	2%

In cryptococcal meningitis, CSF shows high opening pressure and elevated protein. The white cell count may vary according to HIV status

Important for me Less important

Cryptococcus neoformans is correct. This patient has evidence of meningitis given the history of fever, headache and neurological dysfunction. The background of HIV suggests a likelihood of immunosuppression. In immunocompromised patients, it is important to consider alternative organisms that could cause this presentation rather than just those that are most prevalent in the general population. Looking at the lumbar puncture results from this patient, there is a significantly raised opening pressure, elevated protein and low glucose. The white cell count is only marginally raised and is predominantly lymphocytic. It is important to remember that the

white cell count in CSF analysis can vary in patients with HIV depending on the severity of their immunocompromise. *Cryptococcus neoformans* is the most common fungal infection of the CNS in patients with HIV. It causes the lumbar puncture findings seen with this patient's presentation and is, therefore, the most likely causative organism.

Herpes simplex virus is incorrect. Viral meningitis can be seen in immunocompromised and immunocompetent individuals with common responsible viruses including enteroviruses (most common), herpesviruses, mumps and measles. Viral meningitis produces a CSF with elevated protein and a raised white cell count of mostly lymphocyte predominance as seen here. However, it would not cause a raised opening pressure and the CSF glucose is not reduced in patients with viral meningitis making this an unlikely diagnosis.

Listeria monocytes is incorrect. *Listeria monocytes* is an important organism to consider in patients with meningitic symptoms who are over the age of 50 years. Although the majority of bacteria cause neutrophil-dominant leucocytosis in the CSF, *Listeria monocytes* can also cause a lymphocytic predominance. However, the raised opening pressure points away from *Listeria monocytes* as the causative organism.

Neisseria meningitidis is incorrect. This is one of the more common causes of bacterial meningitis. Whilst *Neisseria meningitidis* can cause an elevated opening pressure, the white cell count is generally made up of neutrophils rather than lymphocytes as seen in this scenario.

Streptococcus pneumoniae is incorrect. Like *Neisseria meningitidis*, this is one of the most common causes of bacterial meningitis. However, the lymphocyte-predominant white cell count on CSF analysis makes this organism less likely to be responsible for this patient's presentation.

Discuss (4) | Improve

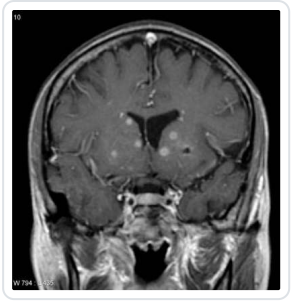
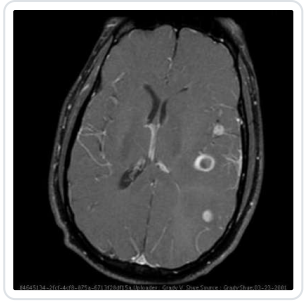
Next question >

HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

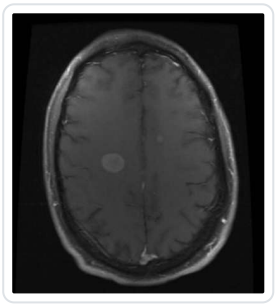
- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination
- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link Report broken link

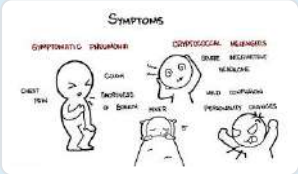
Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

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Report broken media

Score: **15.8%**

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A 29-year-old man presents to his first HIV clinic appointment. He has routine three month HIV tests. He received contact tracing notification last week and was tested and found to be HIV positive. He has just started antiretroviral treatment.

At clinic he mentions that he is due to go to South Africa. He hasn't yet had his travel vaccinations and is worried about what affect the new diagnosis of HIV will have on his travel plans. He is aware that currently immunosuppressed and at risk of infections and is committed to taking his tablets.

Investigations:

Haemoglobin	113 g/L (130-180)
White cell count	6.5 109/L (4.0-11.0)
Neutrophil count	5.4 109/L (1.5-7.0)
Platelet count	170 109/L (150-400)
CD4 count	180 cells/mm3 (600-1500)

Which of the following vaccinations is safe to be given?

- Yellow fever
- BCG
- Oral polio
- Meningococcal C
- Varicella

Submit answer

Reference ranges

Score: **32%**

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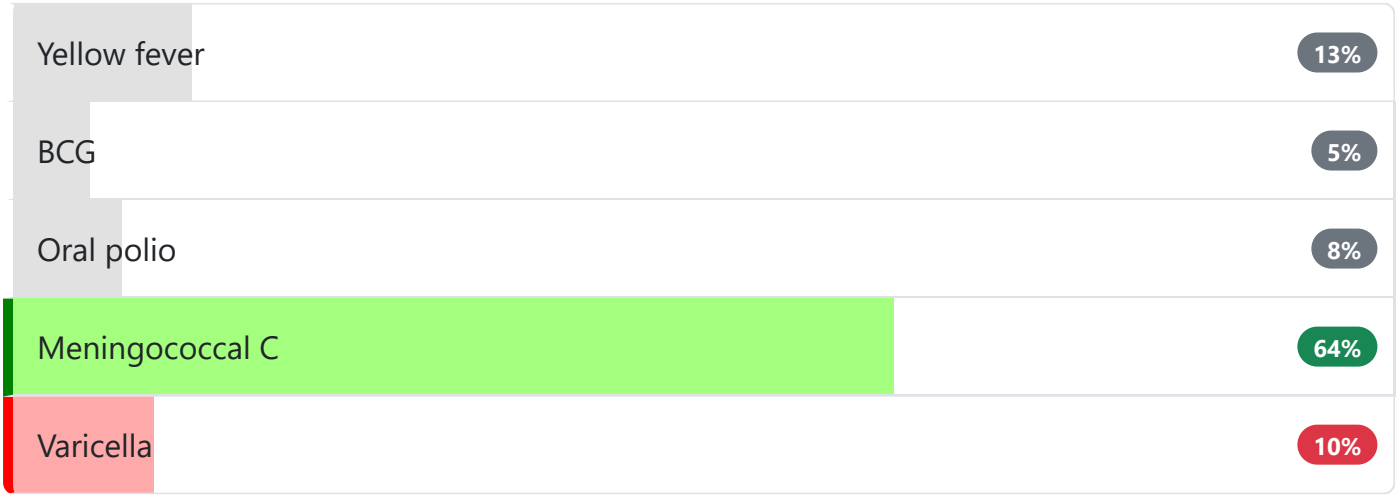
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Platelet count	170 109/L (150-400)
CD4 count	180 cells/mm3 (600-1500)

Which of the following vaccinations is safe to be given?



Given this gentleman recent diagnosis with HIV he is immunocompromised which is reflected by his low CD4 count. He should not be given live vaccines as he is at risk of disseminated infection.

Yellow Fever, BCG, Oral Polio and Varicella are all live vaccines. Meningococcal C is a conjugate vaccine and can be given cautiously to an immunocompromised patient.

Next question

HIV: immunisation

The Department of Health 'Greenbook' on immunisation defers to the British HIV Association for guidelines relating to immunisation of HIV-infected adults

Vaccines that can be used in all HIV-infected adults	**Vaccines that can be used if CD4 > 200**	**Contraindicated in HIV-infected adults**
Hepatitis A Hepatitis B *Haemophilus influenzae* B (Hib) Influenza-parenteral Japanese encephalitis Meningococcus-MenC Meningococcus-ACWY I Pneumococcus-PPV23 Poliomyelitis-parenteral (IPV) Rabies Tetanus-Diphtheria (Td)	Measles, Mumps, Rubella (MMR) Varicella Yellow Fever	Cholera CVD103-HgR Influenza-intranasal Poliomyelitis-oral (OPV) Tuberculosis (BCG)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BHIVA

3 3

2008 HIV immunisation guidelines

Suggest link Report broken link

Score: **15.8%**

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A 34-year-old man with chronic immune thrombocytopenia has not responded to medical therapy and so is scheduled to undergo an elective splenectomy. According to guidelines, he requires the following vaccinations:

- Pneumovax (Pneumococcus), Haemophilus Influenzae, MMR (Measles, Mumps, Rubella), Annual influenza vaccine
- Haemophilus Influenzae, Meningococcus C, Annual influenza vaccine
- Pneumovax (Pneumococcus), Haemophilus Influenzae, Meningococcus C, Annual influenza vaccine
- Pneumovax (Pneumococcus), Haemophilus Influenzae, Meningococcus C
- Meningococcus C, Haemophilus Influenzae, MMR (Measles, Mumps, Rubella), Annual influenza vaccine

Submit answer

Reference ranges

Score: **32%**

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Pneumovax (Pneumococcus), Haemophilus Influenzae, MMR (Measles, Mumps, Rubella), Annual influenza vaccine	4%
Haemophilus Influenzae, Meningococcus C, Annual influenza vaccine	4%
Pneumovax (Pneumococcus), Haemophilus Influenzae, Meningococcus C, Annual influenza vaccine	73%
Pneumovax (Pneumococcus), Haemophilus Influenzae, Meningococcus C	16%
Meningococcus C, Haemophilus Influenzae, MMR (Measles, Mumps, Rubella), Annual influenza vaccine	3%

Risk of infection following splenectomy is with encapsulated bacteria e.g *Streptococcus pneumoniae*. *Haemophilus influenzae* type B, *Neisseria meningitidis*. Therefore patients should be immunised against these infections. MMR vaccination is not required.

Discuss (6) | Improve

Next question

Splenectomy

Following a splenectomy patients are particularly at risk from pneumococcus, Haemophilus, meningococcus and Capnocytophaga canimorsus* infections

Vaccination

- if elective, should be done 2 weeks prior to operation
- Hib, meningitis A & C
- annual influenza vaccination
- pneumococcal vaccine every 5 years

Antibiotic prophylaxis

- penicillin V: unfortunately clear guidelines do not exist of how long antibiotic prophylaxis should be continued. It is generally accepted though that penicillin should be continued for at least 2 years and at least until the patient is 16 years of age, although the majority of patients are usually put on antibiotic prophylaxis for life

Surgical aspects

Indications

- Trauma: 1/4 are iatrogenic
- Spontaneous rupture: EBV
- Hypersplenism: hereditary spherocytosis or elliptocytosis etc
- Malignancy: lymphoma or leukaemia
- Splenic cysts, hydatid cysts, splenic abscesses

Elective splenectomy

- Elective splenectomy is a very different operation from that performed in the emergency setting. The spleen is often large (sometimes massive)
- Most cases can be performed laparoscopically. The spleen will often be macerated inside a specimen bag to facilitate extraction.

Complications

- Haemorrhage (may be early and either from short gastrics or splenic hilar vessels
- Pancreatic fistula (from iatrogenic damage to pancreatic tail)
- Thrombocytosis: prophylactic aspirin
- Encapsulated bacteria infection e.g. *Strep. pneumoniae*, *Haemophilus influenzae* and *Neisseria meningitidis*

Post-splenectomy changes

- Platelets will rise first (therefore in ITP should be given after splenic artery clamped)
- Blood film will change over following weeks, Howell-Jolly bodies will appear
- Other blood film changes include target cells and Pappenheimer bodies
- Increased risk of post-splenectomy sepsis, therefore prophylactic antibiotics and pneumococcal vaccine should be given.

Post-splenectomy sepsis

- Typically occurs with encapsulated organisms
- Opsonisation occurs but then not recognised

*usually from dog bites

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info

5 2

Splenectomy and immunization review

Suggest link

Report broken link

Score: **15.8%**

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A 66 year old lady, originally from Pakistan, has lived in the UK for 10 years. She has type 2 diabetes mellitus which is controlled with metformin only.

For the last few years she has had multiple lumps over her whole body but this hasnt bothered her as it has been there for several years and not got too much worse. She has been seeing the diabetic foot care team due to several traumatic show healing ulcers on her feet.

Over the past few weeks she has been feeling increasingly unwell. She has had a fever, pain in her knees and elbows, sore painful eyes. She also complains of painful swelling in her ring and little fingers on her right hand.

On examination her temperature is 37.8C. Her pulse is 91 beats/minute and her RR is 18 breaths/minute. Her oxygen saturations are 97% on room air.

She has a multiple nodular lesions over her whole body in a symmetrical distribution. Sizes of these nodule range from 2cm - 4cm. These lesions are not painful, in fact with the larger ones she is unable to feel you touching them. On her shins there are several hot painful nodules which are uncomfortable to touch. She has a saddle deformity of her nose. There are several scars on her hands, and an ulcer on the lateral dorsum of her foot. Cardiorespiratory and abdominal examination is essentially normal. Both knees and elbow are slightly erythematous and swollen. She has weakness abducting her little and index finger on her left hand and has no sensation in her left little finger. There is a non-painful 1x4 cm lump two centimetres distal to her left medical epicondyle.

Urinalysis reveals ++ of protein and + of blood.
Blood sugar: 8.8 mmol/L

On the clinical information alone what investigation is most likely to secure the diagnosis?

- Serum cANCA antibody
- Serum dsDNA antibody
- Renal biopsy
- Skin biopsy
- Serum ANA antibody

Submit answer

Reference ranges

Score: **32%**

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A 66 year old lady, originally from Pakistan, has lived in the UK for 10 years. She has type 2 diabetes mellitus which is controlled with metformin only.

For the last few years she has had multiple lumps over her whole body but this hasnt bothered her as it has been there for several years and not got too much worse. She has been seeing the diabetic foot care team due to several traumatic show healing ulcers on her feet.

Over the past few weeks she has been feeling increasingly unwell. She has had a fever, pain in her knees and elbows, sore painful eyes. She also complains of painful swelling in her ring and little fingers on her right hand.

On examination her temperature is 37.8C. Her pulse is 91 beats/minute and her RR is 18 breaths/minute. Her oxygen saturations are 97% on room air.

She has a multiple nodular lesions over her whole body in a symmetrical distribution. Sizes of these nodule range from 2cm - 4cm. These lesions are not painful, in fact with the larger ones she is unable to feel you touching them. On her shins there are several hot painful nodules which are uncomfortable to touch. She has a saddle deformity of her nose. There are several scars on her hands, and an ulcer on the lateral dorsum of her foot. Cardiorespiratory and abdominal examination is essentially normal. Both knees and elbow are slightly erythematous and swollen. She has weakness abducting her little and index finger on her left hand and has no sensation in her left little finger. There is a non-painful 1x4 cm lump two centimetres distal to her left medical epicondyle.

Urinalysis reveals ++ of protein and + of blood.
Blood sugar: 8.8 mmol/L

On the clinical information alone what investigation is most likely to secure the diagnosis?

Serum cANCA antibody	20%
Serum dsDNA antibody	6%
Renal biopsy	9%
Skin biopsy	63%
Serum ANA antibody	2%

With skin lesions and peripheral nerve involvement leprosy should certainly be on a differential diagnosis. Other give away features here are the anaesthetic skin lesions and nerve thickening. The erythema nodosum like lesions and the joint, renal and conjunctival involement indicate a type 2 reaction which occur in patients with lepromatous leprosy (usually following treatment).

Skin biopsy in this case will reveal the diagnosis. This would save her the potentially disastrous consequences of immunosupression.

Discuss (5) Improve

Next question >

Leprosy

Leprosy is a granulomatous disease primarily affecting the peripheral nerves and skin. It is caused by *Mycobacterium leprae*.

Features
- patches of hypopigmented skin typically affecting the buttocks, face, and extensor surfaces of limbs
- sensory loss

The degree of cell mediated immunity determines the type of leprosy a patient will develop.

Low degree of cell mediated immunity → lepromatous leprosy ('multibacillary')
- extensive skin involvement
- symmetrical nerve involvement

High degree of cell mediated immunity → tuberculoid leprosy ('paucibacillary')
- limited skin disease
- asymmetric nerve involvement → hypesthesia
- hair loss

Management
- WHO-recommended triple therapy: rifampicin, dapsone and clofazimine

123

Next question

Textbooks

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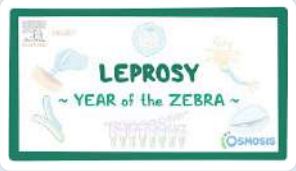
Links

DermNet NZ 6 0

Leprosy

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Media



Leprosy

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A 46 year old Indian gentleman who moved to the UK four years previously presents to the Emergency Department complaining of a numb left foot. He has a background history of type 2 diabetes mellitus, hypertension and gastro-oesophageal reflux disease. His latest haemoglobin A1c (HbA1c) is 6.4%. On examination he has reduced sensation over the left heel and plantar aspect of the foot and six hypoesthesic, hypopigmented patches over both legs. What is the most appropriate management?

- Referral to diabetes specialist clinic for management of peripheral neuropathy
- Dapsone, clofazimine
- Rifamipicin, clofazimine, isoniazid
- Rifamipicin, dapsone, clofazimine
- Dapsone, terbinafine, rifampicin

Submit answer

Reference ranges

Score: **32%**

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Referral to diabetes specialist clinic for management of peripheral neuropathy	3%
Dapsone, clofazimine	15%
Rifamipicin, clofazimine, isoniazid	7%
Rifamipicin, dapsone, clofazimine	65%
Dapsone, terbinafine, rifampicin	9%

The travel history, the distribution of sensory peripheral loss and the pattern of hypoesthesic skin patches are the clues to this patient's diagnosis. Mycobacterium leprae is one of the most common causes of peripheral neuropathy worldwide and has a vast spectrum of presentations from the typical skin manifestations described here to systemic involvement involving the respiratory tract, kidneys, testes and bones.

When there is neurological involvement, the posterior tibial nerve is most commonly affected, (the pattern of sensory loss being described here) however there may equally be involvement of other nerves eg. ulnar, median, lateral popliteal and facial.

Although he may have some underlying diabetic peripheral neuropathy, the pattern of sensory loss would be symmetrical and therefore targeting treatment towards his diabetes will not necessarily help here.

For the treatment of multibacillary leprosy, the WHO have recommended a multi-drug therapy regimen of dapsone, rifampicin and clofazimine for a two year period.

Leprosy

Leprosy is a granulomatous disease primarily affecting the peripheral nerves and skin. It is caused by *Mycobacterium leprae*.

Features

- patches of hypopigmented skin typically affecting the buttocks, face, and extensor surfaces of limbs
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Management

- WHO-recommended triple therapy: rifampicin, dapsone and clofazimine

AI + Q 123

Next question >

Textbooks

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Links

DermNet NZ

6 0

Leprosy

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Media



Leprosy

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A 36-year-old Sri-Lankan woman presents to the GP with a malar rash. On examination she has a erythematous rash that is warm to touch with some underlying oedema over both cheeks and the bridge of her nose.

She reports feeling low in energy over several months and describes a feeling of numbness in her feet and hands.

Investigations:

haemoglobin	125 g/L (115-165)
white cell count	9.4 Ã— 109/L (4.0-11.0)
platelet count	220 Ã— 109/L (150-400)
erythrocyte sedimentation rate	50 mm/1st h (<30)
serum urea	7.0 mmol/L (2.5-7.0)
serum creatinine	105 mol/L (60-110)
serum alanine aminotransferase	17 U/L (5-35)
serum aspartate aminotransferase	26 U/L (1-31)
serum complement C3	80 mg/dL (65-190)
serum complement C4	45 mg/dL (15-50)
serum C-reactive protein	145 mg/L (<10)
anti-double-stranded DNA antibodies	negative
c-ANCA	negative
p-ANCA	negative
antinuclear antibodies	1:20 (negative at 1:20 dilution)

An outpatient rheumatology appointment is arranged and she is started on a course of steroids in the interim. Her symptoms rapidly improve and the rash almost completely resolves however she is left with persistent numbness that appears to be worsening.

What is the most likely underlying diagnosis?

- Granuloma annulare

- Annular psoriasis
- Systemic lupus erythematosus
- Leprosy
- Cutaneous leishmaniasis

Submit answer

Reference ranges

Score: **32%**

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What is the most likely underlying diagnosis?

Granuloma annulare 11%

Annular psoriasis	4%
Systemic lupus erythematosus	12%
Leprosy	61%
Cutaneous leishmaniasis	12%

The diagnosis here is leprosy. Leprosy can present along a spectrum from borderline to leprotamous and can often be mistaken for conditions like psoriasis, SLE and granuloma annulare. The key distinguishing feature is the presence of anaesthetic patches that are present in leprosy but none of the other condition. A full clinical examination is not complete until sensation has been checked over the skin lesions with a microfilament.

The presence of anaesthetic patches should prompt you to look for other hallmarks of disease such as thickened nerves (often easiest to feel are the median nerve in the wrist the common peroneal nerve around the lateral head of the fibula and the ulnar nerve at the elbow.) the loss of hair (in and around lesions and often in the eyebrows) and the presence of neuropathy. Diagnosis is made by demonstrating acid fast bacilli within a cutaneous nerve on microscopy.

The severe skin changes seen with leprosy are not caused by the bacteria themselves but the bodys own immune response towards the bacilli. As a result steroids would improve the physical appearance of skin lesions but at the same time accelerate disease as the bacteria proliferate unhindered. This corresponds to the case outlined above where the patients neurological symptoms worsen yet the skin disease improves on starting steroids.

Discuss (6) | Improve

Next question >

Leprosy ★

Leprosy is a granulomatous disease primarily affecting the peripheral nerves and skin. It is caused by *Mycobacterium leprae*.

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Management

- WHO-recommended triple therapy: rifampicin, dapsone and clofazimine

123

Next question

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Leprosy

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Media



Leprosy

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A 34-year-old man presents to the emergency department with three days of fever, muscle ache, retro-orbital headache and a rash. He has returned from Honduras 5 days ago. He has no past medical history and does not take any regular medications.

Observations:

- Heart rate 101 beats/min
- Oxygen saturations 97% on room air
- Respiratory rate 20 breaths/min
- Temperature 38.5ºC
- Blood pressure 124/82 mmHg

On examination, his face appears flushed. There is a maculopapular rash covering most of his body. There is no splenomegaly. There is no synovitis.

Blood tests:

Hb	121 g/L	Male: (135-180) Female: (115 - 160)
Platelets	89 * 10^9/L	(150 - 400)
WBC	4.4 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	22 mg/L	(< 5)
Bilirubin	14 µmol/L	(3 - 17)
ALP	111 u/L	(30 - 100)
ALT	143 u/L	(3 - 40)
Albumin	34 g/L	(35 - 50)

What is the most likely diagnosis?

- Chikungunya
- Dengue

- Malaria
- Typhoid
- Zika

Submit answer

Reference ranges

Score: **32%**

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CRP	22 mg/L	(< 5)
Bilirubin	14 µmol/L	(3 - 17)
ALP	111 u/L	(30 - 100)
ALT	143 u/L	(3 - 40)
Albumin	34 g/L	(35 - 50)

What is the most likely diagnosis?

Chikungunya	6%
Dengue	86%

Malaria	2%
Typhoid	3%
Zika	3%

Retro-orbital headache, fever, facial flushing, rash, thrombocytopenia in returning traveller → ?dengue

Important for me Less important

Dengue is the correct answer. Dengue virus is transmitted by the Aedes mosquito. It is endemic to Honduras. Facial flushing and a retro-orbital headache are characteristic along with severe myalgia (i.e 'breakbone fever'). Thrombocytopenia and deranged liver function tests are also common.

Chikungunya is incorrect. This can cause fever in the returning traveller and is another virus transmitted by the Aedes mosquito that is found in Honduras. While this condition can cause fever and myalgia, arthralgia and arthritis are almost ubiquitous in this condition and their absence makes this much less likely.

Malaria is incorrect. Malaria is present in Honduras and can cause fever and thrombocytopenia. However, malaria is typically not associated with a maculopapular rash and facial flushing and therefore dengue is more likely.

Typhoid is incorrect. This is another common cause of fever in the returning traveller. This can cause fever, thrombocytopenia and deranged liver function tests. However, it is often associated with abdominal pain and constipation. Additionally, the rash is typically described as 'rose spots' occurring on the lower chest and abdomen rather than a diffuse maculopapular rash and facial flushing. Severe myalgia is also less common. Therefore, dengue is more likely.

Zika is incorrect. This is another virus transmitted by the Aedes mosquito. However, it is not typically associated with a high fever (usually <38ºC) and is often associated with conjunctivitis and itching, which are absent in this case. Dengue is, therefore, more likely.

Discuss (1) Improve

Next question ›

Dengue fever ★

Dengue fever is a viral infection that can progress to viral haemorrhagic fever (other examples include yellow fever, Lassa fever, Ebola).

Aetiology

- dengue virus is a RNA virus of the genus Flavivirus
- transmitted by the *Aedes aegypti* mosquito
- incubation period of 7 days

Clinical features

Patients with dengue fever can be classified as follows:

- dengue fever:
  - without warning signs
  - with warning signs
- severe dengue (dengue haemorrhagic fever)

Dengue fever

- fever
- headache (often retro-orbital)
- myalgia, bone pain and arthralgia ('break-bone fever')
- pleuritic pain
- facial flushing (dengue)
- maculopapular rash
- haemorrhagic manifestations e.g. positive tourniquet test, petechiae, purpura/ecchymosis, epistaxis
- 'warning signs' include:
  - abdominal pain
  - hepatomegaly
  - persistent vomiting
  - clinical fluid accumulation (ascites, pleural effusion)

Severe dengue (dengue haemorrhagic fever)

- this is a form of disseminated intravascular coagulation (DIC) resulting in:
  - thrombocytopenia
  - spontaneous bleeding
- around 20-30% of these patients go on to develop dengue shock syndrome (DSS)

Investigations

- typically blood results
  - leukopenia, thrombocytopenia, raised aminotransferases
- diagnostic tests
  - serology
  - nucleic acid amplification tests for viral RNA
  - NS1 antigen test

Treatment

- entirely symptomatic e.g. fluid resuscitation, blood transfusion etc
- no antivirals are currently available

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 8 14

2011 Dengue fever review

Postgraduate Medical Journal 6 9

Review of dengue fever

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Score: **15.8%**

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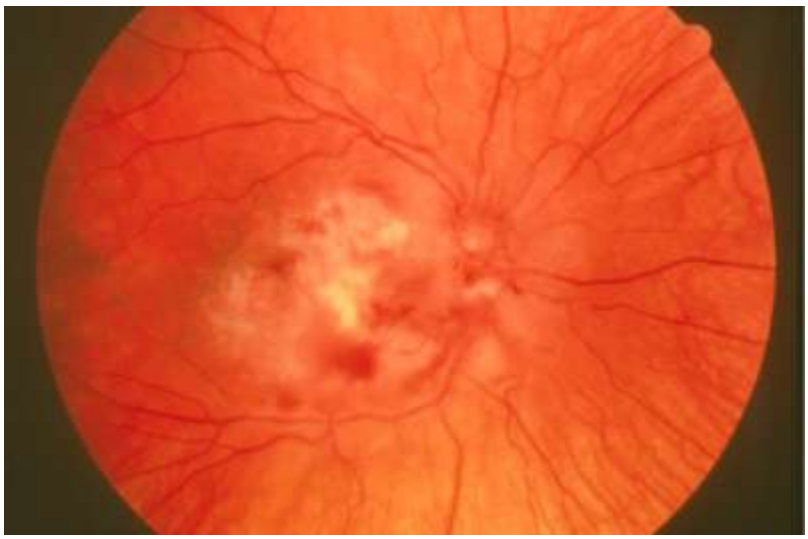
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A 42-year-old man who has recently emigrated to the UK is seen in the HIV clinic. He was diagnosed with HIV 5 years ago and has only sporadically taken anti-retroviral therapy during that period. He complains of blurred vision. Fundoscopy shows the following:



What is the most appropriate treatment?

- Intravenous ganciclovir
- Oral pyrimethamine and sulfadiazine, plus folinic acid
- Intravenous methylprednisolone
- Oral mebendazole
- Oral co-trimoxazole

Submit answer

Reference ranges

Score: **32%**

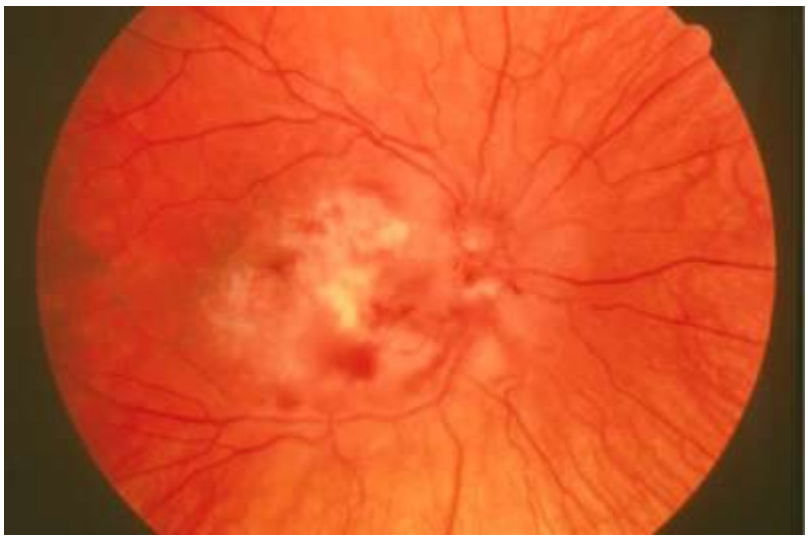
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Back to top

A 42-year-old man who has recently emigrated to the UK is seen in the HIV clinic. He was diagnosed with HIV 5 years ago and has only sporadically taken anti-retroviral therapy during that period. He complains of blurred vision. Fundoscopy shows the following:



What is the most appropriate treatment?

Intravenous ganciclovir	86%
Oral pyrimethamine and sulfadiazine, plus folinic acid	9%
Intravenous methylprednisolone	3%
Oral mebendazole	1%
Oral co-trimoxazole	1%

The fundoscopic image shows changes consistent with cytomegalovirus retinitis.

Discuss (2) | Improve

Next question >

HIV: Cytomegalovirus retinitis

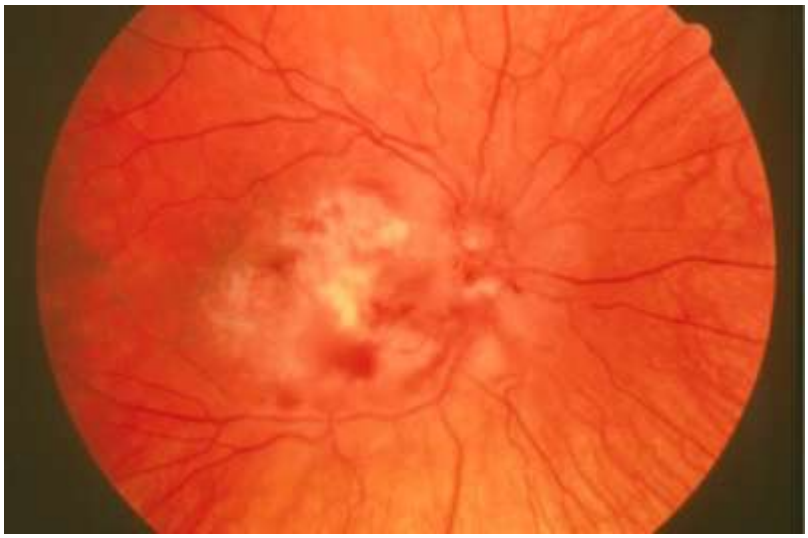
Cytomegalovirus (CMV) retinitis is common, affecting 30-40% of patients who have a CD4 count < 50. Diagnosis is clinical as there are no diagnostic tests

Features

- visual impairment - 'blurred vision' etc

Fundoscopy

- characteristic appearance showing retinal haemorrhages and necrosis
- often called 'pizza' retina



Fundus photograph showing CMV retinitis. Credit: National Eye Institute, National Institutes of Health

Management

- IV ganciclovir
- treatment used to be life-long but new evidence suggests that it may be discontinued once CD4 > 150 after HAART
- alternative: IV foscarnet or cidofovir

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

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Report broken media

Score: **15.8%**

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A 35 year old known HIV positive patient presents with a gradual deterioration in his vision. He states that this has happened over the course of a few weeks. You note from his previous records that he is known to be poorly compliant with his antiretroviral therapy. His most recent CD4 count is 177 cells/mm3. Given the most likely diagnosis, what would be the initial management strategy?

- Oral aciclovir
- Intravenous pentamidine
- Oral pyrimethamine and sulfadiazine, plus folinic acid
- Intravenous ganciclovir
- Oral co-trimoxazole

Submit answer

Reference ranges

Score: **32%**

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Oral aciclovir	3%
Intravenous pentamidine	3%
Oral pyrimethamine and sulfadiazine, plus folinic acid	13%
Intravenous ganciclovir	**78%**
Oral co-trimoxazole	2%

This gentleman has developed cytomegalovirus retinitis treatment of which should be with intravenous ganciclovir or its prodrug oral valganciclovir.

CMV retinitis:

- Presents in those who are HIV +ve with CD4 counts typically <200 cells/mm3.
- Patients may be asymptomatic initially, but tend to present with a gradual deterioration in their visual acuity and visual blurring.
- Typical fundoscopic features described include the 'pizza pie' fundus. Flame hemorrhages may also be seen.
- Treatment is with intravenous ganciclovir or its prodrug oral valganciclovir.

Intravenous pentamidine and oral co-trimoxazole are used to treat *Pneumocystis jirovecii* (formely known as *Pneumocystis carinii*.

Oral pyrimethamine and sulfadiazine, plus folinic acid is used to treat toxoplasmosis.

Discuss (7) | Improve

Next question >

HIV: Cytomegalovirus retinitis

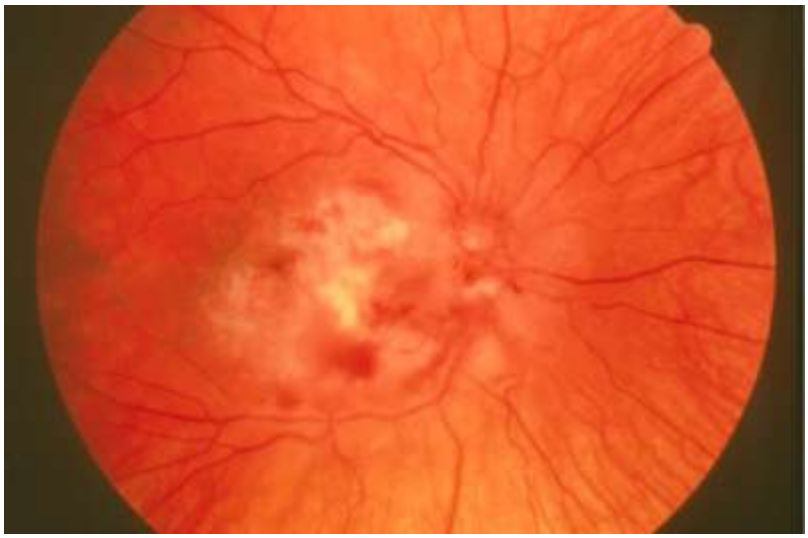
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123

Next question >

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High-yield textbook

Extended textbook

Media



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Armando Hasudungan - YouTube

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Score: **15.8%**

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A 44-year-old woman presents to the emergency department with cough, haemoptysis, and episodes of epistaxis. Additionally, she reports a two week history of fatigue, fever and muscle aches.

On examination, she appears icteric. Coarse crackles are heard on her chest and she has right upper quadrant tenderness. There is tenderness in both her calves and in her lower back.

She is usually fit and well, working as a vet in a nearby town. She is a non-smoker and consumes around 20 units of alcohol a week. At home, she has a pet cat.

Which of the following is most appropriate for this patient?

- Advise her to isolate for the course of her infection as she is at risk of transmission to others
- Advise serology testing is most likely to confirm the diagnosis
- After recovery she should be offered a vaccine as she has occupational risk
- Offer a short course of oral ciprofloxacin
- She should be warned that her cat was likely the source of her infection

Submit answer

Reference ranges

Score: **32%**

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On examination, she appears icteric. Coarse crackles are heard on her chest and she has right upper quadrant tenderness. There is tenderness in both her calves and in her lower back.

She is usually fit and well, working as a vet in a nearby town. She is a non-smoker and consumes around 20 units of alcohol a week. At home, she has a pet cat.

Which of the following is most appropriate for this patient?

Advise her to isolate for the course of her infection as she is at risk of transmission to others	10%
Advise serology testing is most likely to confirm the diagnosis	59%
After recovery she should be offered a vaccine as she has occupational risk	6%
Offer a short course of oral ciprofloxacin	10%
She should be warned that her cat was likely the source of her infection	15%

Serology is the most commonly used test for the diagnosis of leptospirosis, however, antibodies may not be present until after 7 days

Important for me Less important

This patient has occupational and social history that increase the risk of zoonotic infections. She has presented with a febrile illness with components which include liver involvement and a coagulopathy. Together with myalgia classically affecting the calves and lower back this is suggestive of leptospirosis.

The diagnosis is most commonly made by serological testing for specific antibodies. It is difficult to isolate the organism by culture. The greatest diagnostic yield for culture is if it is done in the first 10 days of the illness. After this point, the organisms are only intermittently present in the blood. This patient has had symptoms for at least 2 weeks and therefore culture pick-up rates are now even lower.

An infected individual will shed leptospires in their urine for the period of the illness and for a time

afterwards. Hence there is in theory a transmittable route to others but only in a specific way. Respiratory droplet transmission does not occur and so normal social interactions are safe.

There is no vaccine against leptospirosis available. Therefore, hand hygiene is important in those with occupational risk.

Most cases of leptospirosis are mild and self-resolve without pharmacological management. Where antibiotics are employed, doxycycline or azithromycin are often used. Amoxicillin is also effective.

Leptospirosis is classically associated with rats, which are the most common source of transmission, but other sources include dogs, cattle and pigs.

Discuss (10) | Improve

Next question

Leptospirosis

Leptospirosis is caused by the spirochaete *Leptospira interrogans* (serogroup *L. icterohaemorrhagiae*), classically being spread by contact with infected rat urine.

Epidemiology

- leptospirosis is commonly seen in questions referring to sewage workers, farmers, vets or people who work in an abattoir
- however, on an international level, leptospirosis is far more common in the tropics so should be considered in the returning traveller

Weil's disease should always be considered in high-risk patients with hepatorenal failure

Features

- the early phase is due to bacteraemia and lasts around a week
  - may be mild or subclinical
  - fever
  - flu-like symptoms
  - subconjunctival suffusion (redness)/haemorrhage
- second immune phase may lead to more severe disease (Weil's disease)
  - acute kidney injury (seen in 50% of patients)
  - hepatitis: jaundice, hepatomegaly
  - aseptic meningitis

Investigation

- **serology**: antibodies to *Leptospira* develop after about 7 days
- PCR
- culture
  - growth may take several weeks so limits usefulness in diagnosis
  - blood and CSF samples are generally positive for the first 10 days
  - urine cultures become positive during the second week of illness

Management

- high-dose benzylpenicillin or doxycycline



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Quarterly Journal of Medicine

12 4

Leptospirosis review

Suggest link Report broken link

Media



Leptospira

Osmosis - YouTube 7 0

Report broken media

Score: **15.8%**

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Back to top

A 24-year-old female presented to clinic with symptoms of dysuria and new vaginal discharge. She has been having unprotected sexual intercourse with a new male partner over the past eight weeks. Her nuclear acid amplification (NAAT) test was positive for *Chlamydia* and negative for gonorrhoea infection. She is currently menstruating.

What is the first-line treatment of choice for this infection?

- Amoxicillin
- Azithromycin
- Ceftriaxone
- Doxycycline
- Erythromycin

Submit answer

Reference ranges

Score: **32%**

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What is the first-line treatment of choice for this infection?

Amoxicillin	1%
Azithromycin	13%
Ceftriaxone	7%
Doxycycline	78%
Erythromycin	1%

Chlamydia - treat with doxycycline

Important for me Less important

AI

This patient has a *Chlamydia* infection. We know she is not pregnant as she is currently menstruating. The first-line treatment for *Chlamydia* infection is a 7-day course of doxycycline. Azithromycin was previously preferred as a first-line treatment as it can be given as a one-off dose, which increases compliance. However, recent evidence has raised concern for co-infection with *Chlamydia* and *Mycoplasma genitalium*, with growing resistance to macrolides. Therefore, doxycycline is now recommended as first-line treatment in non-pregnant patients.

Amoxicillin may be considered as a treatment for *Chlamydia* in pregnant patients. However, is not the first-line in this case.

Azithromycin is no longer considered first-line due to growing concerns around resistance. Azithromycin is also considered as a treatment option in pregnant patients with chlamydia infection.

Ceftriaxone is the first-line treatment for patients with *Gonorrhoea*, rather than *Chlamydia* infection. This patient was negative for *Gonorrhoea* infection on NAAT.

Erythromycin may also be considered as a treatment for *Chlamydia* in pregnant patients. However, is not the first-line in this case.

Discuss (1) Improve

Next question >

Chlamydia

Chlamydia is the most prevalent sexually transmitted infection in the UK and is caused by *Chlamydia trachomatis*, an obligate intracellular pathogen. Approximately 1 in 10 young women in the UK have *Chlamydia*. The incubation period is around 7-21 days, although it should be remembered a large percentage of cases are asymptomatic

Features

- asymptomatic in around 70% of women and 50% of men
- women: cervicitis (discharge, bleeding), dysuria
- men: urethral discharge, dysuria

Potential complications

- epididymitis
- pelvic inflammatory disease
- endometritis
- increased incidence of ectopic pregnancies
- infertility
- reactive arthritis
- perihepatitis (Fitz-Hugh-Curtis syndrome)

Investigation

- traditional cell culture is no longer widely used
- nuclear acid amplification tests (NAATs) are now the investigation of choice
- urine (first void urine sample), vulvovaginal swab or cervical swab may be tested using the NAAT technique
- for women: the vulvovaginal swab is first-line
- for men: the urine test is first-line
- *Chlamydia*testing should be carried out two weeks after a possible exposure

Screening

- in England the National *Chlamydia* Screening Programme is open to all men and women aged 15-24 years
- the 2009 SIGN guidelines support this approach, suggesting screening all sexually active patients aged 15-24 years

- relies heavily on opportunistic testing



© Image used on license from PathoPic

Pap smear demonstrating infected endocervical cells. Red inclusion bodies are typical

Management

- doxycycline (7 day course) if first-line
    - this is now preferred to azithromycin due to concerns about *Mycoplasma genitalium*. This infection is often coexistant in patients with *Chlamydia* and there is evidence of rising levels of macrolide resistance, hence why doxycycline is preferred
    - if doxycycline is contraindicated / not tolerated then either azithromycin (1g od for one day, then 500mg od for two days) should be used
- if pregnant then azithromycin, erythromycin or amoxicillin may be used. The SIGN guidelines suggest azithromycin 1g stat is the drug of choice 'following discussion of the balance of benefits and risks with the patient'
- patients diagnosed with *Chlamydia* should be offered a choice of provider for initial partner notification - either trained practice nurses with support from GUM, or referral to GUM
- for men with urethral symptoms: all contacts since, and in the four weeks prior to, the onset of symptoms
- for women and asymptomatic men all partners from the last six months or the most recent sexual partner should be contacted
- contacts of confirmed *Chlamydia* cases should be offered treatment prior to the results of their investigations being known (treat then test)

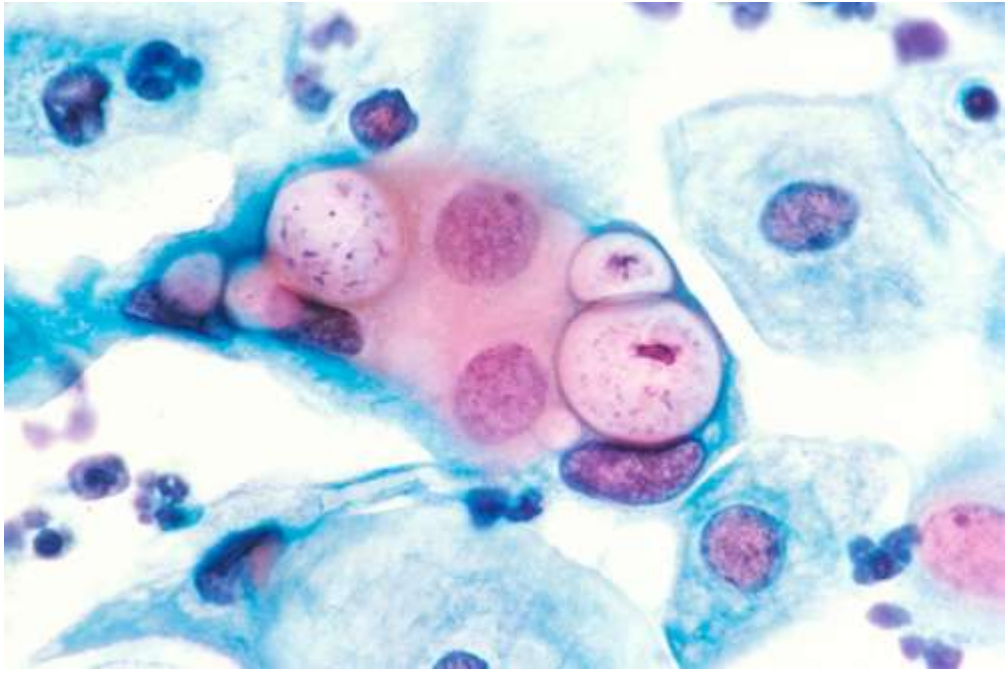


Image sourced from Wikipedia© Image used on license from PathoPic

Another Pap smear demonstrating infected endocervical cells. Stained with H&E

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Association for Sexual Health and HIV

12 11

2018 UK national guideline for the management of infection with Chlamydia trachomatis

Public Health England

13 11

Sexually transmitted infections and chlamydia screening in England, 2014

Clinical Knowledge Summaries

Chlamydia guidelines

18 8

Suggest link Report broken link

Score: **15.8%**

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Back to top

A 23-year-old man presents to a sexual health clinic with a five-day history of dysuria, purulent urethral discharge and urinary frequency. He reports having a long-term partner. He has never previously had a sexual health screening. The patient has no known allergies.

A urine sample is sent for Nucleic Acid Amplification Testing (NAAT) which is negative for chlamydia and gonorrhoea. A urethral swab shows leucocytes. Blood-borne virus testing is negative and cultures of rectal and oral swabs are negative.

What is the most appropriate treatment?

- Antibiotics not indicated
- Intramuscular ceftriaxone
- Oral aciclovir
- Oral doxycycline
- Oral metronidazole

Submit answer

Reference ranges

Score: **32%**

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A urine sample is sent for Nucleic Acid Amplification Testing (NAAT) which is negative for chlamydia and gonorrhoea. A urethral swab shows leucocytes. Blood-borne virus testing is negative and cultures of rectal and oral swabs are negative.

What is the most appropriate treatment?

Antibiotics not indicated	9%
Intramuscular ceftriaxone	28%
Oral aciclovir	2%
Oral doxycycline	48%
Oral metronidazole	13%

Urethritis in a male, negative for *Gonorrhoea* and *Chlamydia* →?*Mycoplasma genitalium*

Important for me Less important

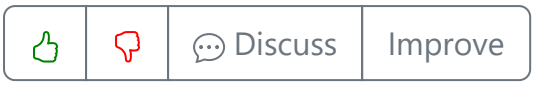
Oral doxycycline is the correct answer. This patient has non-gonococcal urethritis (NGU) which can be caused, by organisms including *Chlamydia trachomatis* and *Mycoplasma genitalium*. NICE recommends doxycycline 100 mg twice a day for 7 days as first-line treatment. If a *Mycoplasma genitalium* NAAT is positive, BASHH recommends oral doxycycline 100mg bd for seven days followed by azithromycin 1g orally as a single dose then 500mg orally once daily for 2 days, where the organism is known to be macrolide-sensitive or where resistance status is unknown.

Antibiotics not indicated is the incorrect answer. Although NAAT testing for chlamydia and gonorrhoea is negative, this patient is symptomatic and requires treatment for urethritis.

Intramuscular ceftriaxone is the incorrect answer. This is the treatment for gonorrhoea and the patient has had a negative gonorrhoea NAAT.

Oral aciclovir is the incorrect answer. This is used to treat herpes simplex virus infection. There is no genital ulceration to suggest herpes infection.

Oral metronidazole is the incorrect answer. This would be the appropriate treatment if *Trichomonas vaginalis* is isolated, which is a cause of NGU. However, metronidazole would not be given first-line for a non-specified NGU.



Next question

Non-gonococcal urethritis

Non-gonococcal urethritis (NGU, sometimes referred to as non-specific urethritis) is a term used to describe the presence of urethritis when a gonococcal bacteria are not identifiable or the initial swab. A typical case would be a male who presented to a GUM clinic with a purulent urethral discharge and dysuria. A swab would be taken in clinic, microscopy performed which showed neutrophils but no Gram-negative diplococci (i.e. no evidence of gonorrhoea). Clearly, this patient requires immediate treatment prior to waiting for the Chlamydia test to come back and hence an initial diagnosis of NGU is made.

No cause is found in around half of cases. Possible causative organisms include:

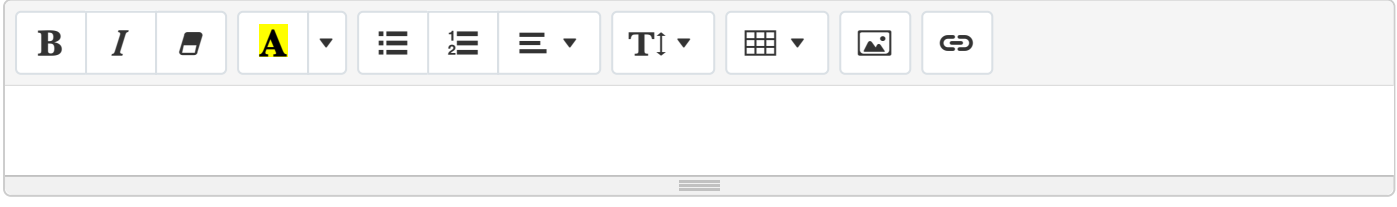
- *Chlamydia trachomatis*
  - most common cause
- *Mycoplasma genitalium*
  - thought to cause more symptoms than *Chlamydia*
- less common causes
  - *Ureaplasma urealyticum*
  - *Trichomonas vaginalis*
  - *Escherichia coli*

Management

- contact tracing
- the BNF and British Association for Sexual Health and HIV (BASHH) both recommend either oral azithromycin or doxycycline



Next question



Textbooks

High-yield textbook

Extended textbook

Links

British Association for Sexual Health and HIV (BASHH)

4 2

2015 UK National Guideline on the management of non-gonococcal urethritis

Suggest link

Report broken link

Score: **15.8%**

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Back to top

A 35-year-old woman presents to the acute medical unit with a 1-week history of worsening headache and fever. Her past medical history is unremarkable. On examination, you note nuchal rigidity and photophobia. A neurological examination is entirely normal. Observations are as follows: heart rate 115 beats per minute, blood pressure 125/75 mmHg, respiratory rate 16 breaths per minute, SpO2 96% (on air), and temperature 38.5ºC.

Blood results are as follows:

Hb	110 g/L	Male: (135-180) Female: (115 - 160)
Platelets	168 * 10^9/L	(150 - 400)
WBC	4.2 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	3.6 mmol/L	(3.5 - 5.0)
Urea	2.4 mmol/L	(2.0 - 7.0)
Creatinine	52 µmol/L	(55 - 120)
CRP	78 mg/L	(< 5)
Glucose	7.8 mmol/L	(0.7-1.0)

HIV testing	HIV-1 antibody detected
HIV viral load	240,890 copies/mL

A lumbar puncture is performed:

Opening pressure	44 cm H2O	(7-18)
WBC count	250 cells/µL (90% lymphocytes)	(0-5)
Glucose	1.8 mmol/L	(0.7-1.0)
Protein	220 mg/dL	(<45)

What is the most likely diagnosis?

- Bacterial meningitis
- CNS lymphoma
- Cerebral toxoplasmosis

- Cryptococcal meningitis
- TB meningitis

Submit answer

Reference ranges

Score: **32%**

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Back to top

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Glucose	1.8 mmol/L	(0.7-1.0)
Protein	220 mg/dL	(<45)

What is the most likely diagnosis?

Bacterial meningitis	3%
CNS lymphoma	3%
Cerebral toxoplasmosis	7%

Cryptococcal meningitis 67%

TB meningitis 21%

In cryptococcal meningitis, CSF shows high opening pressure and elevated protein. The white cell count may vary according to HIV status

Important for me Less important

It is important to note that this patient has a new diagnosis of HIV given the positive HIV-1 antibody result.

Cryptococcal meningitis is correct. Cryptococcal meningitis is the most common fungal CNS infection in patients with HIV. The lymphocytic pleocytosis, low glucose, raised protein, and markedly elevated opening pressure is characteristic of cryptococcal meningitis.

Bacterial meningitis is incorrect. The classic CSF results in bacterial meningitis is a high WBC count with a predominance of neutrophils (>90%).

CNS lymphoma is incorrect. This condition behaves as a space-occupying lesion which would usually be associated with focal neurology.

Cerebral toxoplasmosis is incorrect. Cerebral toxoplasmosis mostly presents as cerebral mass lesions with headache, confusion, fever, lethargy, seizures, cranial nerve palsies, psychomotor changes, hemiparesis and/or ataxia. The absence of neurological symptoms makes this a less likely diagnosis.

TB meningitis is incorrect. Distinguishing TB meningitis from cryptococcal meningitis based on clinical and laboratory features can be a real challenge. TB meningitis also results in a WBC pleocytosis (early neutrophils then lymphocytes), low glucose, and elevated protein. However, in this case, the markedly raised opening pressure favours the diagnosis of cryptococcal meningitis.

Discuss (3) Improve

Next question >

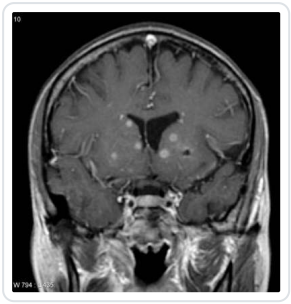
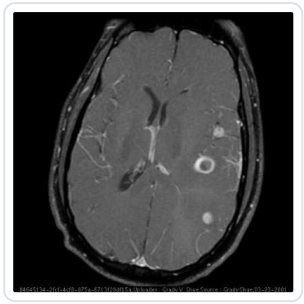
HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

- accounts for around 50% of cerebral lesions in patients with HIV

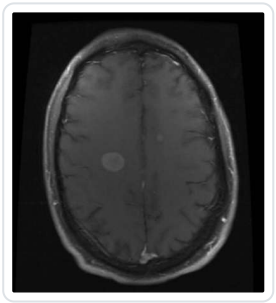
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination
- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link

Report broken link

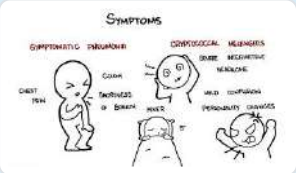
Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

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Report broken media

Score: **15.8%**

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A 49-year-old man presents to the emergency department with fever and dry cough for the last week. He also complains of myalgia and headaches which have been hampering his work on his farm. These headaches are variable in location, but usually worse behind his eyes. He is an ex-smoker, with a 15 pack-year history. His alcohol intake is minimal and he has no significant past medical history of note.

On examination, he has coarse crackles in his right lung base. His abdomen is soft and non-tender. There is no rash. Heart sounds are normal, with no murmurs heard.

Initial investigations are done and a chest x-ray is requested.

Hb	146 g/L	Male: (135-180) Female: (115 - 160)
Platelets	452 * 10^9/L	(150 - 400)
WBC	14.8 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.7 mmol/L	(3.5 - 5.0)
Urea	6.9 mmol/L	(2.0 - 7.0)
Creatinine	118 µmol/L	(55 - 120)
CRP	83 mg/L	(< 5)
Bilirubin	17 µmol/L	(3 - 17)
ALP	122 u/L	(30 - 100)
ALT	84 u/L	(3 - 40)
Î³GT	66 u/L	(8 - 60)
Albumin	39 g/L	(35 - 50)

What is the most appropriate treatment option?

- Albendazole
- Amoxicillin
- Azithromycin
- Co-trimoxazole

Doxycycline

Submit answer

Reference ranges

Score: **32%**

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CRP	83 mg/L	(< 5)
Bilirubin	17 µmol/L	(3 - 17)
ALP	122 u/L	(30 - 100)
ALT	84 u/L	(3 - 40)
Î³GT	66 u/L	(8 - 60)
Albumin	39 g/L	(35 - 50)

What is the most appropriate treatment option?

Albendazole	7%
Amoxicillin	6%
Azithromycin	15%
Co-trimoxazole	5%

Doxycycline 68%

Farmer, fever, transaminitis ?Q fever

Important for me Less important

This patient is a farmer who has presented with evidence of pneumonia combined with a transaminitis. His occupation should make you think about potential zoonotic infections, which cause this presentation. Some examples of zoonotic infections include brucellosis, cryptosporidiosis, orf and Q fever.

Q fever is caused by *Coxiella burnetii*, a rickettsia. The source of infection is typically an abattoir (cattle/sheep). It may be inhaled from infected dust. It's features include a prodromal illness, atypical pneumonia and a transaminitis, as in this case. Treatment is most effective if given early, and doxycycline is the antibiotic of choice.

Amoxicillin may be used in cases of community-acquired pneumonia to cover typical organisms. However the transaminitis and occupation in the question point to a more atypical presentation.

Azithromycin is a potential treatment option for typhoid fever, which is more common in returning travellers from Asia. It often causes a prodromal illness and can affect the liver. In this case, there is no mention of travel and other features of typhoid such as a 'rose-spots' rash and bradycardia are absent.

Co-trimoxazole is a combination antibiotic, consisting of trimethoprim and sulfamethoxazole. It is the treatment of choice for pneumocystis pneumonia. However, this is rare in immunocompetent patients, classically occurring in those with HIV and a CD4 count below 200 cells/mm^3.

Discuss (5) Improve

Next question >

Q fever

Q fever is caused by *Coxiella burnetii*, a rickettsia. The source of infection is typically an abattoir, cattle/sheep or it may be inhaled from infected dust

Features

- typically prodrome: fever, malaise
- causes pyrexia of unknown origin
- transaminitis
- atypical pneumonia
- endocarditis (culture-negative)

Management

- doxycycline

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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Raymond is a 35-year-old man presenting with fevers. He had just returned from his overseas trip 7 days ago and he started experiencing fevers 3 days after his return. This was accompanied by joint pain along his wrists and ankles. The pain was so severe that he struggled to walk and lift objects with his hands due to the pain. Paracetamol and ibuprofen did not help the pain. He also felt generally fatigues over this time.

Raymond has recently travelled to Africa and India over the last 2 months. He states that he had gotten all of the recommended vaccinations before travelling. He notes that he had taken Malarone as his malaria prophylaxis throughout the trip. He had applied mosquito repellent during the 1st month of the trip but had forgotten to replenish this during the 2nd month of the trip.

Examination revealed swollen joints that were hot to touch but not overtly erythematous over both his wrist and ankle. An erythematous rash is also noted across the body with no clear discrete lesions identified. This was not pruritic in nature. Pyrexia was noted at 38.4ºC.

Blood tests results were as follow:

Hb	130 g/L	Male: (135-180) Female: (115 - 160)
Platelets	90 * 10^9/L	(150 - 400)
WBC	5.0 * 10^9/L	(4.0 - 11.0)
Neuts	3.7 * 10^9/L	(2.0 - 7.0)
Lymphs	0.7 * 10^9/L	(1.0 - 3.5)
Mono	0.5 * 10^9/L	(0.2 - 0.8)
Eosin	0.1 * 10^9/L	(0.0 - 0.4)

Na^+	137 mmol/L	(135 - 145)
K^+	4.0 mmol/L	(3.5 - 5.0)
Urea	6.0 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
Bilirubin	10 µmol/L	(3 - 17)
ALP	50 u/L	(30 - 100)
ALT	23 u/L	(3 - 40)
Î³GT	56 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

Three thick and thin blood films were negative.

What is the likely cause for his fevers?

- ○ Chikungunya fever
- ○ Dengue fever
- ○ Leptospirosis
- ○ Malaria
- ○ Yellow fever

Submit answer

Reference ranges

Score: **32%**

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Raymond is a 35-year-old man presenting with fevers. He had just returned from his overseas trip 7 days ago and he started experiencing fevers 3 days after his return. This was accompanied by joint pain along his wrists and ankles. The pain was so severe that he struggled to walk and lift objects with his hands due to the pain. Paracetamol and ibuprofen did not help the pain. He also felt generally fatigues over this time.

Raymond has recently travelled to Africa and India over the last 2 months. He states that he had gotten all of the recommended vaccinations before travelling. He notes that he had taken Malarone as his malaria prophylaxis throughout the trip. He had applied mosquito repellent during the 1st month of the trip but had forgotten to replenish this during the 2nd month of the trip.

Examination revealed swollen joints that were hot to touch but not overtly erythematous over both his wrist and ankle. An erythematous rash is also noted across the body with no clear discrete lesions identified. This was not pruritic in nature. Pyrexia was noted at 38.4ºC.

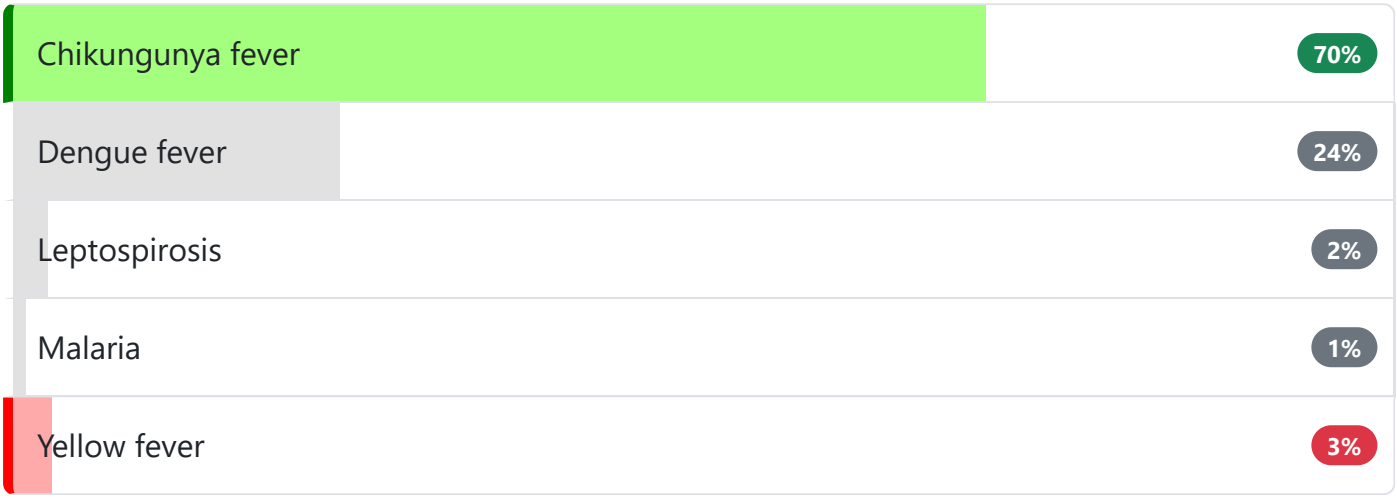
Blood tests results were as follow:

Hb	130 g/L	Male: (135-180) Female: (115 - 160)
Platelets	90 * 10^9/L	(150 - 400)
WBC	5.0 * 10^9/L	(4.0 - 11.0)
Neuts	3.7 * 10^9/L	(2.0 - 7.0)
Lymphs	0.7 * 10^9/L	(1.0 - 3.5)
Mono	0.5 * 10^9/L	(0.2 - 0.8)
Eosin	0.1 * 10^9/L	(0.0 - 0.4)

Na^+	137 mmol/L	(135 - 145)
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ALT	23 u/L	(3 - 40)
Î³GT	56 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

Three thick and thin blood films were negative.

What is the likely cause for his fevers?



Chikungunya can present with debilitating joint pain

Important for me Less important

The history of travel to Africa and India with hints that the patient may not have engaged with adequate mosquito protection places him at risk for mosquito-borne illness. The combination of rash, fevers and significant joint pains makes chikungunya fever the most likely culprit. Its vector is the *Aedes* mosquito and is endemic within Africa and Asia. It can cause issues like lymphopenia and thrombocytopenia on blood tests. Treatment is supportive.

Dengue fever is less likely as it is not commonly associated with significant joint pains. Instead, it is usually associated with myalgic pains. It will also cause a circulatory collapse in its severe presentation, which is not common in chikungunya.

Yellow fever is also predominantly endemic in Africa. It is less likely in this case given the absence of any significant liver abnormalities. Also, hepatomegaly can be picked up during examination in these patients.

Leptospirosis is predominantly spread by rodents. There is no clear risk of exposure to its vector and it is also not usually associated with joint pains. It is more commonly seen with myalgia and as the disease progresses, liver and renal involvement start to become a prominent feature.

Lastly, malaria is a common disease in both these countries. However, anaemia and jaundice would be expected. Besides, the 3x negative blood films are reassuring and useful to rule out malaria as the underlying cause.

Discuss (5) Improve

Next question

Chikungunya

Alphavirus disease caused by infected mosquitoes. Areas affected are Africa, Asia and Indian subcontinent but in recent years there has been seen in a few cases in Southern Europe. Tanzania had the first reported case.

Symptoms: Prominent symptoms are severe joint pain and abrupt onset of high fever. Other symptoms include general flu-like illness of muscle ache, headache, and fatigue. The disease shares its symptoms with dengue but tends to have more joint pain which can be debilitating. A rash may develop as with other viral illness and swelling of the joints in not uncommon.

Treatment: Relief of symptoms. No specific treatment.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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Back to top

A previously well 28-year-old man presents with shortness of breath and abdominal discomfort. He reports a dry cough for the previous 10 days. He works full time as a management consultant. He is a non-smoker and drinks approximately 20 units of alcohol a week. He went on a stag-do to Prague with a bunch of friends 2 weeks ago. In the last few days, he has noticed a widespread skin rash which he describes as lots of pink rings around a pale centre.

Bloods on admission:

Na^+	128 mmol/l
K^+	3.7 mmol/l
Urea	8.2 mmol/l
Creatinine	150 µmol/l

Chest x-ray: Diffuse reticular infiltrates and small right-sided pleural effusion.

What is the most likely causative organism?

- *Staphylococcus aureus*
- *Pneumocystis jirovecii*
- *Mycoplasma pneumoniae*
- *Streptococcus pneumoniae*
- *Haemophilus influenzae*

Submit answer

Reference ranges

Score: **32%**

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Bloods on admission:

Na^+	128 mmol/l
K^+	3.7 mmol/l
Urea	8.2 mmol/l
Creatinine	150 µmol/l

Chest x-ray: Diffuse reticular infiltrates and small right-sided pleural effusion.

What is the most likely causative organism?

Staphylococcus aureus	1%
Pneumocystis jirovecii	3%
Mycoplasma pneumoniae	91%
Streptococcus pneumoniae	3%
Haemophilus influenzae	2%

Stereotypical history of mycoplasma pneumonia: worsening flu-like symptoms and a dry cough. Erythema multiforme is noted on examination

Important for me Less important

This patient has community-acquired pneumonia. Whilst *Streptococcus pneumoniae* and *Haemophilus influenzae* are the commonest causative organisms they tend to present with a shorter history. The longer duration of symptoms and unusual features of abdominal pain, dry cough and hyponatraemia should alert you to an atypical organism of which *Mycoplasma pneumoniae* is one of the commonest. The patient also describes erythema multiforme which is seen with *Mycoplasma pneumonia*.

Mycoplasma pneumoniae occurs in epidemics every three to four years and typically affects younger people. It tends to have an insidious onset with flu-like symptoms and atypical features. Hyponatraemia is often seen. Cold agglutinins can cause an autoimmune haemolytic anaemia. Diagnosis is confined with Mycoplasma serology. Treatment is with clarithromycin or a tetracycline or fluoroquinolone.

Staphylococcal pneumonia may complicate influenza infection and is seen most frequently in the elderly and in intravenous drug users or patients with underlying disease. It can result in a cavitating pneumonia.

Pneumocystis jirovecii is seen in immunocompromised patients. It typically presents with exertional breathlessness and patients are seen to desaturate on walking. Whilst a HIV test should be considered in the diagnostic workup of this patient, it is not the most likely diagnosis in this case.

Discuss (5) | Improve

Next question >

Mycoplasma pneumoniae

Mycoplasma pneumoniae is a cause of atypical pneumonia which often affects younger patients. It is associated with a number of characteristic complications such as erythema multiforme and cold autoimmune haemolytic anaemia. Epidemics of *Mycoplasma pneumoniae* classically occur every 4 years. It is important to recognise atypical pneumonia as it may not respond to penicillins or cephalosporins due to it lacking a peptidoglycan cell wall.

Features

- the disease typically has a prolonged and gradual onset
- flu-like symptoms classically precede a dry cough
- bilateral consolidation on x-ray
- complications may occur as below

Complications

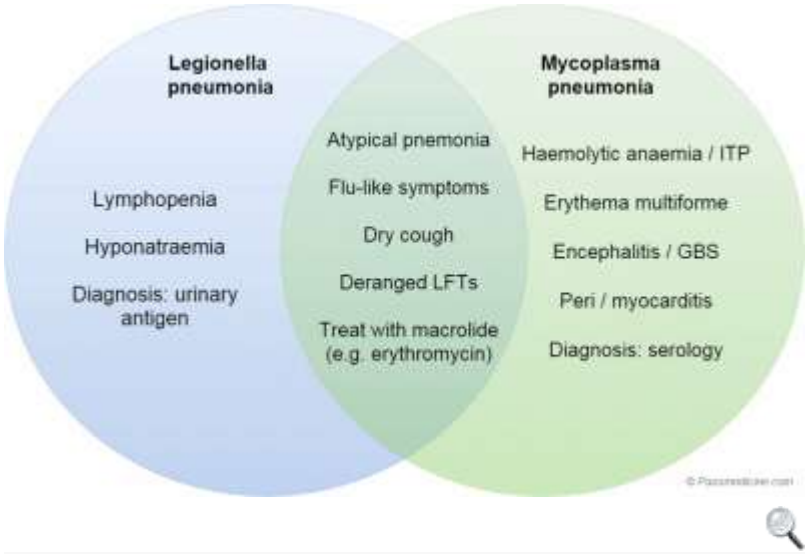
- cold agglutins (IgM): may cause an haemolytic anaemia, thrombocytopenia
- erythema multiforme, erythema nodosum
- meningoencephalitis, Guillain-Barre syndrome and other immune-mediated neurological diseases
- bullous myringitis: painful vesicles on the tympanic membrane
- pericarditis/myocarditis
- gastrointestinal: hepatitis, pancreatitis
- renal: acute glomerulonephritis

Investigations

- diagnosis is generally by Mycoplasma serology
- positive cold agglutination test → peripheral blood smear may show red blood cell agglutination

Management

- doxycycline or a macrolide (e.g. erythromycin/clarithromycin)



Comparison of Legionella and Mycoplasma pneumonia

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ

11 6

Erythema multiforme

Suggest link Report broken link

Media



Mycoplasma pneumoniae

Osmosis - YouTube 1 0

Report broken media

Score: **15.8%**

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Back to top

A 45-year-old man attends the general outpatient clinic having recently returned from hiking holiday in the Highlands of Scotland. Following his return, he describes how he has been feeling a bit unwell. He states that he has been experiencing general muscle aches, fatigue and a mild headache. On examination, they have a red papule on the posterior aspect of their calf with a patch of clear skin and a further red ring. It is non-tender and there is no warmth.

Further examination is unremarkable.

What is the most appropriate initial action?

- Discharge with reassurance and worsening advice
- ELISA antibody test *B. Burgdorferi*
- Oral doxycycline
- PCR test
- Skin biopsy

Submit answer

Reference ranges

Score: **32%**

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Further examination is unremarkable.

What is the most appropriate initial action?

Discharge with reassurance and worsening advice	2%
ELISA antibody test *B. Burgdorferi*	11%
Oral doxycycline	85%
PCR test	1%
Skin biopsy	1%

Lyme disease can be diagnosed clinically if erythema migrans is present → give antibiotics

Important for me Less important

The best initial option is to treat them empirically for Lyme disease. They have been out walking in an endemic area for Lyme disease and have features of early disease (erythema migrans and non-specific viral features). Early treatment for suspected Lyme disease is appropriate in people who present with erythema migrans and other features of infection. Specific investigations for Lyme disease may be appropriate if the clinical presentation is delayed.

There is no requirement for further investigations as they have features of Lyme disease so a skin biopsy, ELISA PCR test is inappropriate as these would not change the management. Similarly, as they have presented with erythema migrans and there is good reason to suspect Lyme disease, discharging with no treatment is also inappropriate.

Next question

Lyme disease

Lyme disease is caused by the spirochaete *Borrelia burgdorferi* and is spread by ticks.

Early features (within 30 days)

- erythema migrans
  - 'bulls-eye' rash is typically at the site of the tick bite
  - typically develops 1-4 weeks after the initial bite but may present sooner
  - usually painless, more than 5 cm in diameter and slowlly increases in size
  - present in around 80% of patients.
- systemic features
  - headache
  - lethargy
  - fever
  - arthralgia





Later features (after 30 days)

- cardiovascular
  - heart block
  - peri/myocarditis
- neurological
  - facial nerve palsy
  - radicular pain
  - meningitis



Investigation

- NICE recommend that Lyme disease can be diagnosed clinically if erythema migrans is present
  - erythema migrans is therefore an indication to start antibiotics

- enzyme-linked immunosorbent assay (ELISA) antibodies to *Borrelia burgdorferi* are the first-line test
    - if negative and Lyme disease is still suspected in people tested within 4 weeks from symptom onset, repeat the ELISA 4-6 weeks after the first ELISA test. If still suspected in people who have had symptoms for 12 weeks or more then an immunoblot test should be done
    - if positive or equivocal then an immunoblot test for Lyme disease should be done

Management of asymptomatic tick bites

- tick bites can be a relatively common presentation to GP practices, and can cause significant anxiety
- if the tick is still present, the best way to remove it is using fine-tipped tweezers, grasping the tick as close to the skin as possible and pulling upwards firmly. The area should be washed following.
- NICE guidance does not recommend routine antibiotic treatment to patients who've suffered a tick bite

Management of suspected/confirmed Lyme disease

- doxycycline if early disease. Amoxicillin is an alternative if doxycycline is contraindicated (e.g. pregnancy)
    - people with erythema migrans should be commenced on antibiotic without the need for further tests
- ceftriaxone if disseminated disease
- Jarisch-Herxheimer reaction is sometimes seen after initiating therapy: fever, rash, tachycardia after first dose of antibiotic (more commonly seen in syphilis, another spirochaetal disease)



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries — 11 13

Lyme disease: management

NICE — 11 10

2018 Lyme disease guidelines

Dermnet NZ — 12 5

Lyme disease

Suggest link — Report broken link

Media



Lyme disease

Osmosis - YouTube 4 1

Report broken media

Score: **15.8%**

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A 30-year-old female presented to the emergency department with a 5-day history of fever, malaise, myalgia, joint pains, and headache. She thought she was recovering but over the past 2 days, her eyes had started to turn yellow. She had no past medical history and was on no regular medications. She had recently returned from Brazil 8 days ago where she had spent 2 weeks participating in many outdoor sporting events, including a forest marathon and wild swimming in lakes. She denied having a regular partner but reported that she had unprotected sexual intercourse on her last night in Brazil. She does not remember being bitten by insects.

On examination, she looked unwell. Her observations were as follows:

- temperature 39.1°C
- respiratory rate 22/min
- heart rate 109bpm
- blood pressure 105/67mmHg
- SpO2 98% on room air

She had scleral icterus and mild tender hepatomegaly. A faint petechial rash could be seen on her torso, hands, and feet. Blood tests results were as follows:

Hb	101 g/L	Male: (135-180) Female: (115 - 160)
Platelets	83 * 10^9/L	(150 - 400)
WBC	15 * 10^9/L	(4.0 - 11.0)
Neuts	11 * 10^9/L	(2.0 - 7.0)
Lymphs	3 * 10^9/L	(1.0 - 3.5)
Mono	0.6 * 10^9/L	(0.2 - 0.8)
Eosin	0.4 * 10^9/L	(0.0 - 0.4)
Bilirubin	80 µmol/L	(3 - 17)
ALP	231 u/L	(30 - 100)
ALT	265 u/L	(3 - 40)
AST	524 u/L	(5 - 40)

- HBsAg - negative
- Anti-HBs - positive
- Anti-HBc - positive

Which test is most likely to reveal the diagnosis?

- Skin scrapings for *Leishmania braziliensis* culture and PCR
- Serum measurement of HBeAg
- Urine microscopy for detection of *Schistosoma haematobium*
- Treponema pallidum particle agglutination (TPPA) assay
- *Leptospira interrogans* serum serology

Submit answer

Reference ranges

Score: **32%**

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Back to top

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Mono	0.6 * 10^9/L	(0.2 - 0.8)
Eosin	0.4 * 10^9/L	(0.0 - 0.4)
Bilirubin	80 µmol/L	(3 - 17)
ALP	231 u/L	(30 - 100)
ALT	265 u/L	(3 - 40)
AST	524 u/L	(5 - 40)

- HBsAg - negative
- Anti-HBs - positive
- Anti-HBc - positive

Which test is most likely to reveal the diagnosis?

Skin scrapings for *Leishmania braziliensis* culture and PCR	12%
Serum measurement of HBeAg	10%
Urine microscopy for detection of *Schistosoma haematobium*	11%
Treponema pallidum particle agglutination (TPPA) assay	5%
Leptospira interrogans serum serology	62%

Serology is the most commonly used test for the diagnosis of leptospirosis, however, antibodies may not be present until after 7 days

Important for me Less important

Leptospirosis is caused by zoonotic spirochete bacteria, spread via contact with infected animal urine. Animals such as rodents act as reservoirs of infection, shedding the bacteria in their urine which subsequently enter mucous membranes and broken skin. Freshwater swimming and other watersports are associated with leptospirosis infection. After an incubation period of 7 - 14 days, the disease presents in a biphasic fashion: acute fever, malaise, headache, and abdominal pain followed by severe systemic upset such as renal failure, pulmonary haemorrhage, and hepatic failure. Thrombocytopenia is also seen (suggested mechanisms include poor production by bone marrow and immune-mediated platelet destruction). Serology is the most commonly used test for the diagnosis of leptospirosis, however, antibodies may not be present until after 7 days.

Leishmania brasiliensis is the unicellular parasite responsible for cutaneous and mucosal leishmaniasis. Endemic in South America, this disease presents with skin and mucosal ulcerative lesions and is spread by the sandfly. Leishmaniasis is diagnosed by detecting Leishmania parasites (or DNA) in tissue specimens. This patient had no such skin lesions and no history of insect bites.

This patient's hepatitis serology demonstrates that she has been exposed to hepatitis B (positive anti-HBc and anti-HBs) but has cleared the virus (negative HBsAg). Hepatitis B e antigen (HBeAg) is an indicator of viral replication and correlates with hepatitis B virus infectivity. This would typically be measured if the patient had active hepatitis B. As this patient is HepBsAg negative, this test is not necessary.

Schistosoma haematobium is a parasitic fluke endemic to areas of Africa. The intermediate host is the freshwater snail; transmission risk is increased by swimming in such bodies of water. Whilst acute infection can cause a transient febrile illness (Katayama syndrome) the history typically includes pruritis (swimmers itch) with eosinophilia. Schistosomiasis is primarily a chronic disease with *Schistosoma haematobium* causing urological manifestations such as cystitis or obstructive uropathy. Examination of the urine for ova is the primary method used to diagnose suspected *Schistosoma haematobium* infections.

Treponema pallidum a sexually transmitted bacterial spirochete responsible for causing syphilis.

Primary syphilis is characterised by a painless self-limiting genital ulcer (chancre) and lymphadenopathy occurring 10-90 days after sexual transmission. Secondary syphilis occurs following the haematogenous spread of the bacteria and can present with a variety of symptoms including a widespread rash, adenopathy, arthralgia, hepatitis, and alopecia. This classically presents 4-8 weeks after the presentation of the chancre. Whilst this diagnosis should be considered given the patient's sexual history, there is no mention of a genital lesion in the question stem, and the very short period between the sexual encounter (8 days prior) and the onset of her symptoms is not typical of secondary syphilis. The Treponema pallidum particle agglutination (TPPA) assay is one of many tests that may be used to detect specific anti-treponemal antibodies in serum. This is usually only requested after a positive non-treponemal screening test in the diagnostic workup of syphilis.

Discuss (3) | Improve

Next question >

Leptospirosis ★

Leptospirosis is caused by the spirochaete *Leptospira interrogans* (serogroup *L. icterohaemorrhagiae*), classically being spread by contact with infected rat urine.

Epidemiology

- leptospirosis is commonly seen in questions referring to sewage workers, farmers, vets or people who work in an abattoir
- however, on an international level, leptospirosis is far more common in the tropics so should be considered in the returning traveller

Weil's disease should always be considered in high-risk patients with hepatorenal failure

Features

- the early phase is due to bacteraemia and lasts around a week
  - may be mild or subclinical
  - fever
  - flu-like symptoms
  - subconjunctival suffusion (redness)/haemorrhage
- second immune phase may lead to more severe disease (Weil's disease)
  - acute kidney injury (seen in 50% of patients)
  - hepatitis: jaundice, hepatomegaly
  - aseptic meningitis

Investigation

- **serology**: antibodies to *Leptospira* develop after about 7 days

- PCR
- culture
  - growth may take several weeks so limits usefulness in diagnosis
  - blood and CSF samples are generally positive for the first 10 days
  - urine cultures become positive during the second week of illness

Management

- high-dose benzylpenicillin or doxycycline

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Quarterly Journal of Medicine 12 4

Leptospirosis review

Suggest link Report broken link

Media



Leptospira

Osmosis - YouTube 7 0

Report broken media

Score: **15.8%**

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Back to top

A 25-year-old sexually active woman presents with abnormal vaginal discharge and burning during urination. A nucleic acid amplification test (NAAT) for sexually transmitted infections is performed.

NAAT	positive for *Chlamydia trachomatis*

She has no allergies, is using oral contraceptives, and is not pregnant or breastfeeding.

What antibiotic is most appropriate in this case?

- Azithromycin
- Ceftriaxone
- Doxycycline
- Erythromycin
- Trimethoprim

Submit answer

Reference ranges

Score: **32%**

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A 25-year-old sexually active woman presents with abnormal vaginal discharge and burning during urination. A nucleic acid amplification test (NAAT) for sexually transmitted infections is performed.

NAAT	positive for *Chlamydia trachomatis*

She has no allergies, is using oral contraceptives, and is not pregnant or breastfeeding.

What antibiotic is most appropriate in this case?

Azithromycin	10%
Ceftriaxone	6%
Doxycycline	**82%**
Erythromycin	1%
Trimethoprim	0%

Chlamydia - treat with doxycycline

Important for me Less important

National Institute for Health and Care Excellence (NICE) guidance states that **doxycycline** is now the first-line treatment for anyone over the age of 13 diagnosed with *Chlamydia* who is not pregnant or breastfeeding. This is therefore the correct answer.

Azithromycin is the second-line treatment recommended by NICE for treating *Chlamydia* in patients for whom doxycycline is contraindicated or not tolerated.

Ceftriaxone is used in the treatment of *Gonorrhoea* but is not recommended for treating *Chlamydia*.

Erythromycin is a third-line agent recommended by NICE for treating *Chlamydia* in patients for whom both doxycycline and azithromycin are contraindicated or not tolerated.

Trimethoprim is indicated in treating urinary tract infections in some populations however is not used in the treatment of *Chlamydia*.

Discuss | Improve

Next question >

Chlamydia

Chlamydia is the most prevalent sexually transmitted infection in the UK and is caused by *Chlamydia trachomatis*, an obligate intracellular pathogen. Approximately 1 in 10 young women in the UK have *Chlamydia*. The incubation period is around 7-21 days, although it should be remembered a large percentage of cases are asymptomatic

Features

- asymptomatic in around 70% of women and 50% of men
- women: cervicitis (discharge, bleeding), dysuria
- men: urethral discharge, dysuria

Potential complications

- epididymitis
- pelvic inflammatory disease
- endometritis
- increased incidence of ectopic pregnancies
- infertility
- reactive arthritis
- perihepatitis (Fitz-Hugh-Curtis syndrome)

Investigation

- traditional cell culture is no longer widely used
- nuclear acid amplification tests (NAATs) are now the investigation of choice
- urine (first void urine sample), vulvovaginal swab or cervical swab may be tested using the NAAT technique
- for women: the vulvovaginal swab is first-line
- for men: the urine test is first-line
- *Chlamydia*testing should be carried out two weeks after a possible exposure

Screening

- in England the National *Chlamydia* Screening Programme is open to all men and women aged 15-24 years
- the 2009 SIGN guidelines support this approach, suggesting screening all sexually active patients aged 15-24 years

- relies heavily on opportunistic testing



© Image used on license from PathoPic

Pap smear demonstrating infected endocervical cells. Red inclusion bodies are typical

Management

- doxycycline (7 day course) if first-line
    - this is now preferred to azithromycin due to concerns about *Mycoplasma genitalium*. This infection is often coexistant in patients with *Chlamydia* and there is evidence of rising levels of macrolide resistance, hence why doxycycline is preferred
    - if doxycycline is contraindicated / not tolerated then either azithromycin (1g od for one day, then 500mg od for two days) should be used
- if pregnant then azithromycin, erythromycin or amoxicillin may be used. The SIGN guidelines suggest azithromycin 1g stat is the drug of choice 'following discussion of the balance of benefits and risks with the patient'
- patients diagnosed with *Chlamydia* should be offered a choice of provider for initial partner notification - either trained practice nurses with support from GUM, or referral to GUM
- for men with urethral symptoms: all contacts since, and in the four weeks prior to, the onset of symptoms
- for women and asymptomatic men all partners from the last six months or the most recent sexual partner should be contacted
- contacts of confirmed *Chlamydia* cases should be offered treatment prior to the results of their investigations being known (treat then test)

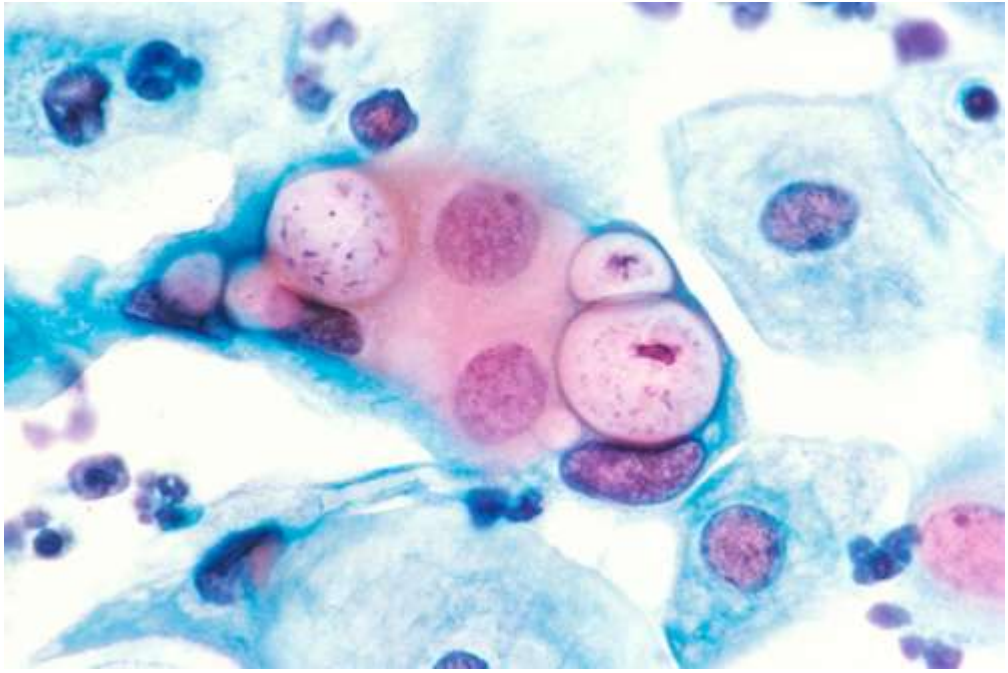


Image sourced from Wikipedia© Image used on license from PathoPic

Another Pap smear demonstrating infected endocervical cells. Stained with H&E

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Association for Sexual Health and HIV

12 11

2018 UK national guideline for the management of infection with Chlamydia trachomatis

Public Health England

13 11

Sexually transmitted infections and chlamydia screening in England, 2014

Clinical Knowledge Summaries

Chlamydia guidelines

18 8

Suggest link Report broken link

Score: **15.8%**

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A 47-year-old male presents to the emergency department with joint pains and rash developing over 48 hours. He describes feeling weak and tired with headaches, myalgia, and lumbar back pain.

Examination reveals painful tender distal inter-phalangeal (DIP) joints and tender wrists in a symmetric distribution. He tells you the pain is worse in the morning and improves throughout the day. He has an erythematous maculopapular rash involving his torso and upper limbs with multiple insect bites. During your assessment, he develops a fever of 39.7°C. His chest is clear on auscultation. There is no palpable lymphadenopathy. He denies any nausea, vomiting, or diarrhoea.

His medical history includes asthma, obstructive sleep apnoea, and depression. He tells you he returned from a family holiday in Mexico 5 days earlier.

Blood tests:

Hb	148g/L	Male: (135-180) Female: (115 - 160)
Platelets	98* 10^9/L	(150 - 400)
WBC	7.8* 10^9/L	(4.0 - 11.0)
Neuts	6.8* 10^9/L	(2.0 - 7.0)
Lymphs	0.2* 10^9/L	(1.0 - 3.5)
Mono	0.6* 10^9/L	(0.2 - 0.8)
Eosin	0.2* 10^9/L	(0.0 - 0.4)
Urea	6.8mmol/L	(2.0 - 7.0)
Creatinine	128 µmol/L	(55 - 120)
CRP	24mg/L	(< 5)

What is the most likely cause of this man's presentation?

- Dengue
- Rheumatoid arthritis
- Malaria
- Chikungunya

Psoriatic arthritis

Submit answer

Reference ranges

Score: **32%**

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Eosin	0.2* 10^9/L	(0.0 - 0.4)
Urea	6.8mmol/L	(2.0 - 7.0)
Creatinine	128 µmol/L	(55 - 120)
CRP	24mg/L	(< 5)

What is the most likely cause of this man's presentation?

Dengue	28%
Rheumatoid arthritis	1%
Malaria	2%
Chikungunya	66%

Psoriatic arthritis 4%

Chikungunya can present with debilitating joint pain

Important for me Less important

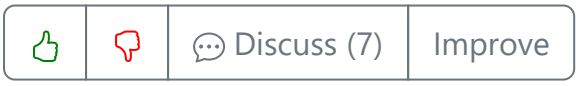
Chikungunya can present with debilitating joint pain. A rash develops in up to 75% of patients. The most common symptoms include fevers and malaise. It can be difficult to differentiate from dengue early in the disease. Two significant differences may help distinguish between them; 1) In chikungunya, rashes appear on the torso and the arms while in dengue, they appear on the arms and the face. 2) Joint pain in chikungunya is experienced in the hands, wrists, feet, and legs, while in dengue it is experienced in the shoulders and the knees.

Dengue is an important differential diagnosis. It is a mosquito-borne tropical disease caused by the dengue virus. Symptoms include a high fever, headache, vomiting, joint pains, and a characteristic skin rash. It may be indistinguishable from chikungunya early in the disease.

Rheumatoid arthritis is a symmetric polyarthritis involving large and small joints. This is unlikely given the acute presentation, and rheumatoid arthritis is not typically associated with fevers or rash.

Malaria should be considered in any patient with fevers and non-specific symptoms travelling from an endemic area. It may present with fevers, malaise, diaphoresis, headaches, cough, arthralgia. Chikungunya is more likely in our patient as it more commonly presents with joint pains and rash.

Psoriatic arthritis is associated with small joint involvement (DIPs) and a characteristic scaly rash. The acute presentation and fevers suggest an infectious cause.



Next question

Chikungunya

Alphavirus disease caused by infected mosquitoes. Areas affected are Africa, Asia and Indian subcontinent but in recent years there has been seen in a few cases in Southern Europe. Tanzania had the first reported case.

Symptoms: Prominent symptoms are severe joint pain and abrupt onset of high fever. Other symptoms include general flu-like illness of muscle ache, headache, and fatigue. The disease shares its symptoms with dengue but tends to have more joint pain which can be debilitating. A rash may develop as with other viral illness and swelling of the joints in not uncommon.

Treatment: Relief of symptoms. No specific treatment.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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A 45-year-old man is known to be HIV positive but has a long history of poor compliance with anti-retroviral medication. He presents to the Emergency Department with a 4-day history of headache and vomiting. On examination, he has neck stiffness but no rash. He has a temperature of 38.6 ºC and is confused. A CT head is unremarkable. A lumbar puncture is performed. Examination of CSF with India ink is positive.

CD4 count	60 cells per mm^3	(500 - 1500)

What additional finding from the lumbar puncture would be most in keeping with the diagnosis?

- Normal glucose
- Normal protein
- Opening pressure of 28 cm H2O
- Presence of organism-specific antibodies in the CSF
- Raised white cell count with predominantly neutrophils

Submit answer

Reference ranges

Score: **32%**

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CD4 count	60 cells per mm^3	(500 - 1500)

What additional finding from the lumbar puncture would be most in keeping with the diagnosis?

Normal glucose	11%
Normal protein	5%
Opening pressure of 28 cm H2O	63%
Presence of organism-specific antibodies in the CSF	13%
Raised white cell count with predominantly neutrophils	7%

In cryptococcal meningitis, CSF shows high opening pressure and elevated protein. The white cell count may vary according to HIV status

Important for me Less important

This patient has cryptococcal meningitis. *Cryptococcus neoformans* is an encapsulated yeast that is found in the environment and is a common cause of meningitis in patients with HIV with a CD4 count of less than 100. Examination of the CSF with India ink is positive in 70-90% of cases of HIV-related cryptococcal meningitis.

Opening pressure of 28 cm H2O is correct. All patients with suspected cryptococcal meningitis should have manometry performed as intracranial pressure is often significantly raised. Patients can require serial lumbar punctures to lower intracranial pressure, or may even require neurosurgical devices such as extra-ventricular drains or ventriculoperitoneal shunts to manage their intracranial pressure.

Normal glucose is incorrect. CSF glucose is generally low in cryptococcal meningitis, although it can sometimes be normal.

Normal protein is incorrect. CSF protein is usually elevated in cryptococcal meningitis.

Presence of organism-specific antibodies in the CSF is incorrect. Antibodies to *Cryptococcus neoformans* are not used in diagnosis, either in the blood or CSF. Cryptococcal antigen (CrAg), however, is a useful test and can be carried out on both blood and CSF. A positive CSF CrAg has a 98% sensitivity for cryptococcal meningitis. High CSF CrAg is a poor prognostic marker.

Raised white cell count with predominantly neutrophils is incorrect. The white cell count is usually elevated, but the predominant cell type would be lymphocytes. Patients with HIV may not have an elevated CSF white cell count, and this is a poor prognostic marker.

Discuss | Improve

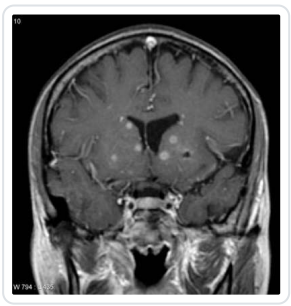
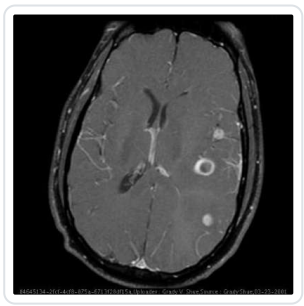
Next question >

HIV: neurocomplications

Focal neurological lesions

Toxoplasmosis

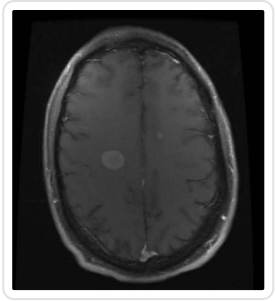
- accounts for around 50% of cerebral lesions in patients with HIV
- constitutional symptoms, headache, confusion, drowsiness
- CT: usually single or multiple ring enhancing lesions, mass effect may be seen
- management: sulfadiazine and pyrimethamine

Primary CNS lymphoma

- accounts for around 30% of cerebral lesions
- associated with the Epstein-Barr virus
- CT: single or multiple homogenous enhancing lesions
- treatment generally involves steroids (may significantly reduce tumour size), chemotherapy (e.g. methotrexate) + with or without whole brain irradiation. Surgical may be considered for lower grade tumours





Differentiating between toxoplasmosis and lymphoma is a common clinical scenario in HIV patients. It is clearly important given the vastly different treatment strategies. The table below gives some general differences. Please see the Radiopaedia link for more details.

Toxoplasmosis	**Lymphoma**
Multiple lesions Ring or nodular enhancement Thallium SPECT negative	Single lesion Solid (homogenous) enhancement Thallium SPECT positive

Tuberculosis

- much less common than toxoplasmosis or primary CNS lymphoma
- CT: single enhancing lesion

Generalised neurological disease

Encephalitis

- may be due to CMV or HIV itself
- HSV encephalitis but is relatively rare in the context of HIV
- CT: oedematous brain

Cryptococcus

- most common fungal infection of CNS
- headache, fever, malaise, nausea/vomiting, seizures, focal neurological deficit
- CSF
  - high opening pressure
  - elevated protein
  - reduced glucose
  - normally a lymphocyte predominance but in HIV white cell count many be normal
  - India ink test positive
- CT: meningeal enhancement, cerebral oedema
- meningitis is typical presentation but may occasionally cause a space-occupying lesion

Progressive multifocal leukoencephalopathy (PML)

- widespread demyelination

- due to infection of oligodendrocytes by JC virus (a polyoma DNA virus)
- symptoms, subacute onset : behavioural changes, speech, motor, visual impairment
- CT: single or multiple lesions, no mass effect, don't usually enhance. MRI is better - high-signal demyelinating white matter lesions are seen

AIDS dementia complex

- caused by HIV virus itself
- symptoms: behavioural changes, motor impairment
- CT: cortical and subcortical atrophy



Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia

11 4

Toxoplasmosis vs lymphoma

Suggest link Report broken link

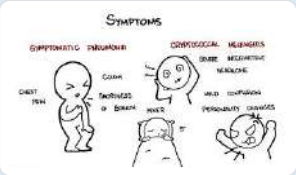
Media



Opportunistic Infections and AIDS-defining illnesses - CD4+ cell count, malignancy, treatment

Armando Hasudungan - YouTube

6 1



Cryptococcus neoformans infection

Armando Hasudungan - YouTube

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Report broken media

Score: **15.8%**

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Back to top

A 27-year-old male attends the emergency department with a 5-day history of fevers, myalgia, and headaches. He is a non-smoker and has no previous medical history.

On examination he appears dyspnoeic, his oxygen saturations are 92% with a respiratory rate of 24/minute. Heart sounds are normal and the abdomen is soft,non-tender with no organomegaly. A chest x-ray reveals subtle bilateral interstitial infiltrates.

Blood tests:

Hb	153g/L	Male: (135-180) Female: (115 - 160)
Platelets	380* 10^9/L	(150 - 400)
WBC	15.1* 10^9/L	(4.0 - 11.0)
Na^+	138mmol/L	(135 - 145)
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Creatinine	96 µmol/L	(55 - 120)
Bilirubin	12 µmol/L	(3 - 17)
ALP	2721u/L	(30 - 100)
ALT	354u/L	(3 - 40)
Î³GT	468u/L	(8 - 60)
Albumin	37g/L	(35 - 50)

He works on a dairy farm with his father, who was recently treated in hospital for pneumonia. His father tested negative for *COVID19* while in hospital. He tells you he has had limited social contacts in the last number of weeks to protect his ageing father from COVID19.

Which of the following is the most likely cause for his presentation?

- Q fever
- *Mycoplasma pneumonia*
- *COVID19*
- Brucellosis

Psittacosis

Submit answer

Reference ranges

Score: **32%**

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Which of the following is the most likely cause for his presentation?

Q fever	67%
Mycoplasma pneumonia	7%
COVID19	1%
Brucellosis	22%

Psittacosis 3%

Farmer, fever, transaminitis ?Q fever

Important for me Less important

Q fever is the correct answer. Q fever *(Coxiella Burnetti)* is a zoonotic infection most commonly found in farmers and abattoir workers. It presents as a non-specific flu-like illness with fevers, headaches, fatigue, and myalgia. Q fever may present as pneumonia and is commonly associated with transaminitis. Occasionally it may present with endocarditis or other localized infections such as bone and joint infections or vascular infections (infected aneurysm). Q fever should be suspected in patients who present with symptoms and have risk factors (working closely with animals, exposure to infected dust, lab work).

Mycoplasma pneumonia may cause a similar presentation. Mycoplasma pneumonia is characterized by a non-specific flu-like illness with patchy infiltrates on chest x-ray, However, mycoplasma pneumonia is rarely associated with significant transaminitis and an alternative diagnosis should be considered.

COVID19 is less likely as he denies any potential exposure.

Brucellosis is incorrect. Brucellosis is associated with dairy farming and may have a similar presentation to q fever. However, brucellosis is more frequently associated with international travel. A minority of cases are transmitted within the UK and Ireland.

Psittacosis is typically associated with transmission from infected birds ('Parrot fever'). Transmission from dairy cattle has been reported but this is a much less likely occurrence. This patient does not disclose any contact with infected birds.

Discuss (8) Improve

Next question >

Q fever

Q fever is caused by *Coxiella burnetii*, a rickettsia. The source of infection is typically an abattoir, cattle/sheep or it may be inhaled from infected dust

Features

- typically prodrome: fever, malaise
- causes pyrexia of unknown origin
- transaminitis
- atypical pneumonia

- endocarditis (culture-negative)

Management
- doxycycline

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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Back to top

A 45-year-old man presents with a 5-day history of fever and headache, followed by the onset of rash and drowsiness on the day of presentation. He has been working as a counsellor in a refugee camp in Ethiopia and returned to the UK 7 days ago. The camp is over capacity and the living conditions are very crowded. The facilities in the camp for personal hygiene and washing clothes are severely limited for both volunteers and residents. There are reports that many people in the camp have also become unwell.

On examination, he is mildly confused and drowsy with a Glasgow Coma Score of 13 (eyes 3, voice 4, motor 6.) He is visibly jaundiced with a palpable liver edge. He has a maculopapular rash on his trunk. A chest x-ray reveals bilateral patchy pulmonary infiltrates.

What antibiotic would be the first line in the management of this patient?

- Benzylpenicillin
- Ciprofloxacin
- Doxycycline
- Metronidazole
- Rifampicin

Submit answer

Reference ranges

Score: **32%**

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What antibiotic would be the first line in the management of this patient?

Benzylpenicillin	19%
Ciprofloxacin	12%
Doxycycline	62%
Metronidazole	4%
Rifampicin	3%

Doxycycline is typically used in the management of typhus

Important for me Less important

This patient has epidemic typhus, which is caused by *Rickettsia prowazekii*, which is a small bacterium transmitted by the bites of the human body louse. Epidemic typhus mainly occurs in outbreaks in conditions of severe upheaval and overcrowding, such as in conflict situations or after natural disasters. It is the most severe infection of the typhus group and has a mortality of 20-50% if untreated. It presents with abrupt onset of headache and fever, followed by the development of rash at day 5, and is commonly complicated by neurological symptoms or multi-organ failure.

The first-line treatment for all rickettsial infections is **doxycycline** . This includes infections in both the spotted fever group and the typhus group. Other management in the outbreak setting would include doxycycline prophylaxis for healthcare workers and mass delousing.

Benzylpenicillin is incorrect. Beta-lactam-based antibiotics are not effective against rickettsial infections. Benzylpenicillin can be used for bacterial meningitis, but this diagnosis is less likely in this patient who also has liver and respiratory involvement.

Ciprofloxacin is incorrect. Ciprofloxacin is not routinely used for the treatment of typhus. It could be considered for treating severe cholera, but this is unlikely in this patient given the lack of diarrhoea. (Doxycycline can also be used to treat cholera in addition to typhus).

Metronidazole is incorrect. Metronidazole is active against anaerobes but does not have any activity against rickettsia.

Rifampicin is incorrect. Rifampicin has some activity against rickettsia, but outcomes are significantly worse than with doxycycline so it is therefore not used in treatment.

Discuss (9) | Improve

Next question >

Typhus ★

Typhus is a group of rickettsial diseases transmitted between hosts by arthropods. Examples include

- endemic typhus
  - caused by *Rickettsia typhi*
  - reservoir/vector: fleas on rats
  - occurs throughout the world, typically in warm costal regions
  - relative bradycardia
- epidemic typhus
  - caused by *Rickettsia prowazekii*
  - reservoir/vector: body louse
  - more common in central and eastern Africa, central and South America
- scrub typhus
  - caused by *Orientia tsutsugamushi*
  - reservoir/vector: harvest mites on humans or rodents
  - more common in Asia
  - black eschar at site of original inoculation
  - relative bradycardia despite fever
- spotted fever
  - caused by Rickettsia spotted fever group
  - spread by ticks
  - examples include Rocky Mountain spotted fever

Common features

- fever, headache, malaise
- rash
  - typically maculopapular
  - begins on the trunk and spreads to the extremities
- later complications
  - meningoencephalitis

Management
- doxycycline

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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Back to top

A 35-year-old farmer presents to the Emergency Department with a one week history of lethargy and subjective pyrexia. Over the past 24 hours he has developed right sided pleuritic chest pain which brought him to hospital.

He denies any past medical history and had been systemically well prior to this. He denies any recent overseas travel.

Examination reveals right sided coarse crackles in the lungs, there is no abnormality heard in the left side of the lung.

His observations revealed heart rate 105 beats per minute, oxygen saturation 96% in room air, temperature 38.1ºC, respiratory rate 18 breaths per minute, blood pressure 145/95 mmHg.

An ECG was performed which revealed sinus tachycardia and his chest radiograph revealed right sided consolidation.

His blood results taken on admission were as follows:

Hb	140 g/L	Male: (135-180) Female: (115 - 160)
Platelets	500 * 10^9/L	(150 - 400)
WBC	16 * 10^9/L	(4.0 - 11.0)
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Urea	6 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
CRP	159 mg/L	(< 5)

Bilirubin	16 µmol/L	(3 - 17)
ALP	78 u/L	(30 - 100)
ALT	80 u/L	(3 - 40)
Î³GT	55 u/L	(8 - 60)
Albumin	40 g/L	(35 - 50)

What is the most likely diagnosis?

- ○ *Mycoplasma pneumoniae* ×
- ○ Influenza ×
- ○ Legionnaires disease ×
- ○ Pulmonary embolism ×
- ○ Q fever ×

Submit answer

Reference ranges

Score: **32%**

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Albumin	40 g/L	(35 - 50)

What is the most likely diagnosis?

Mycoplasma pneumoniae	19%
Influenza	3%
Legionnaires disease	8%
Pulmonary embolism	1%
Q fever	70%

Farmer, fever, transaminitis ?Q fever

Important for me Less important

This patient has Q fever. His possible exposure to sheep given that he is a farmer, pneumonia and mild transaminitis point towards Q fever as a diagnosis.

Influenza is a possibility as it may present with pyrexia and tachycardia along with respiratory symptoms, the clue in this history if that the patient is a farmer, thereby making Q fever more likely.

Legionnaires disease is usually spread through infected air conditioning units in aeroplanes or hotels. This patient has no history of travel.

Mycoplasma would classically present with hyponatraemia in the context of pneumonia, it is not usually caused by exposure to sheep or cattle. which this patients has most likely been exposed to.

A pulmonary embolism could be a differential with pleuritic chest pain and tachycardia, however the likely exposure to sheep, high temperature and consolidation on the chest radiograph makes Q fever more likely.

Discuss (6) Improve

Q fever

Q fever is caused by *Coxiella burnetii*, a rickettsia. The source of infection is typically an abattoir, cattle/sheep or it may be inhaled from infected dust

Features

- typically prodrome: fever, malaise
- causes pyrexia of unknown origin
- transaminitis

- atypical pneumonia
- endocarditis (culture-negative)

Management

- doxycycline

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

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